



Severe Mpox Proctitis Complicated by Bowel Obstruction

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ABSTRACT

Mpox is a rare infection caused by the zoonotic orthopoxvirus. We present the case of a 44-year-old man with HIV and a history of kidney transplant who presented with mpox and developed proctitis-associated bowel obstruction, urinary retention, and eosinophilia. Our case highlights potential gastrointestinal manifestations of severe mpox infection.

KEYWORDS: bowel obstruction; mpox; proctitis

INTRODUCTION

Mpox typically presents as a self-limited prodrome of fevers, vesicular lesions, and lymphadenopathy. Vesicular lesions are painful and can lead to complications, including systemic inflammation and secondary bacterial infections. Gastrointestinal manifestations of mpox include nausea, vomiting, diarrhea,odynophagia, anorexia, and rectal pain/proctitis.¹ Tecovirimat (also known as TPOXX or ST-246) is an antiviral treatment option for certain populations at high risk of severe mpox infection, such as immunosuppressed patients.^{2–4} This case highlights severe gastrointestinal complications of mpox, including proctitis-associated bowel obstruction.

CASE REPORT

A 44-year-old man with a history of HIV infection and prior kidney transplant because of HIV nephropathy presented to the emergency department with a 1-week history of diarrhea, dysuria, urinary retention, and new vesicular lesions throughout his body. He reported adherence to his antiretroviral therapy for HIV (abacavir, lamivudine, and dolutegravir) and transplant immunosuppressant therapy (mycophenolate mofetil, tacrolimus, and prednisone). The patient had sexual encounters with men 20 years earlier, but he reported a monogamous relationship with his wife and engaged in anal intercourse using sexual devices. He had traveled in the Southeast region of the United States a few weeks before presentation. He did not report any pet or animal exposure. On physical examination, well-circumscribed umbilicated lesions were noted on the patient's lips, face, abdomen, and perianal area (Figure 1). Other than mild suprapubic tenderness, his abdomen was not tender or distended.

Serologic workup was supportive of leukocytosis at $11.2 \times 10^9/L$ (reference range, $3.4\text{--}9.6 \times 10^9/L$), and polymerase chain reaction of oral lesions was positive for non-variola orthopoxvirus, suggestive of mpox. Urinalysis was negative for urinary tract infection. The patient started a 2-week course of tecovirimat and was discharged with a Foley catheter for his urinary retention. Two days later, the patient returned to the hospital because of severe proctalgia, diarrhea, progressive urinary retention, hematuria, and decreased oral intake.

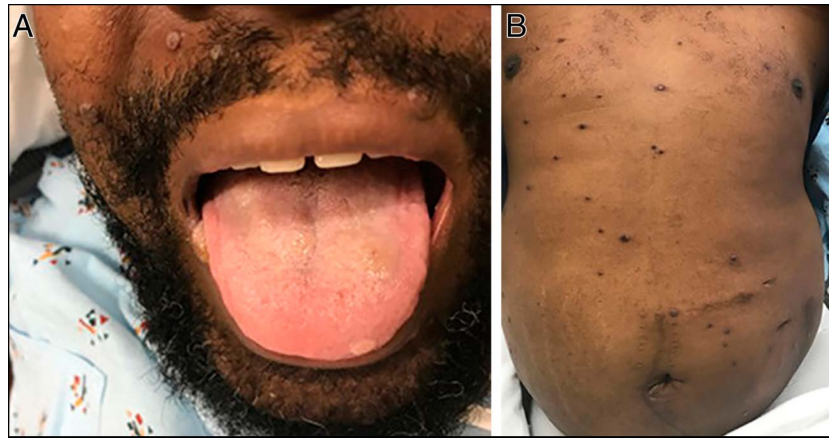


Figure 1. Facial lesions. (A) Numerous maculopapular lesions on the patient's face, including on the lips, buccal mucosa, and tongue. (B) Painful, firm, well-circumscribed, vesicular lesions with central umbilication in various stages of desquamation over the patient's abdomen and trunk.

Laboratory workup supported persistent leukocytosis at $12.4 \times 10^9/L$ with new eosinophilia at $1.03 \times 10^9/L$ and 8.3% (reference range, $0.03\text{--}0.48 \times 10^9/L$, 1.0%–3.0%). Urinalysis showed 4 white blood cells per high-powered field (HPF) (reference range, 0–3/HPF) and 7 red blood cells per HPF (reference range, 0–2/HPF). CD4 count was 145 cells/ μL (reference range 365–1,437 cells/ μL), previously at 149 cells/ μL a year before, and HIV viral load was undetectable. Physical examination was significant for previous vesicular lesions with new diffuse abdominal tenderness. Abdominal and pelvic computed tomography without contrast was supportive of severe proctitis (Figure 2) with associated lymphadenopathy in the mesorectum extending superiorly into the inferior mesenteric artery chain and retroperitoneum (Figure 2).

The patient was admitted to the hospital for fluid support and pain management. Rectal swab for gonorrhea, chlamydia, herpes simplex virus, syphilis, lymphogranuloma venereum, and cytomegalovirus was negative. Stool culture was positive for superimposed enteropathogenic *Escherichia coli* infection.

Tecovirimat was continued, and intravenous ceftriaxone was started for enteropathogenic *E. coli*. On hospital day 3, the patient developed new-onset fever and worsening abdominal pain. The care team continued to withhold oral nutrition. On physical examination, his abdomen was rigid and tympanic on percussion, but without peritoneal signs. Repeat abdominal and pelvic computed tomography without contrast revealed severe proctitis with new distention of the rectosigmoid junction, raising suspicion of developing colonic obstruction related to rectal wall edema (Figure 3). There was also a separate new central abdominal small bowel dilation with air fluid levels suggesting possible developing small bowel obstruction (Figure 3) believed to be associated with adhesions secondary to the history of renal transplant; however, no discrete transition point was identified.

Pain management included intravenous hydromorphone 0.5 mg as needed for days 1–3, oral oxycodone 5 mg as needed for days 4–5, and then acetaminophen as needed until discharge. A colorectal surgeon was consulted for the bowel obstructions and

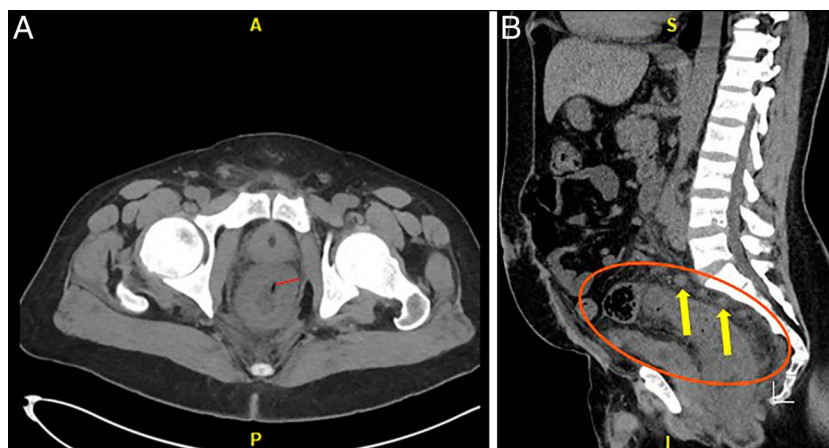


Figure 2. Severe proctitis on abdominal and pelvic computed tomography. (A) Rectal wall thickening measuring up to 20.4 mm (red line). (B) Severe rectal wall thickening with associated perirectal inflammatory stranding (red oval) and inferior mesenteric artery chain adenopathy (yellow arrows). Abrupt transition to normal wall thickness at the rectosigmoid junction. A, anterior; I, inferior; P, posterior; S, superior.

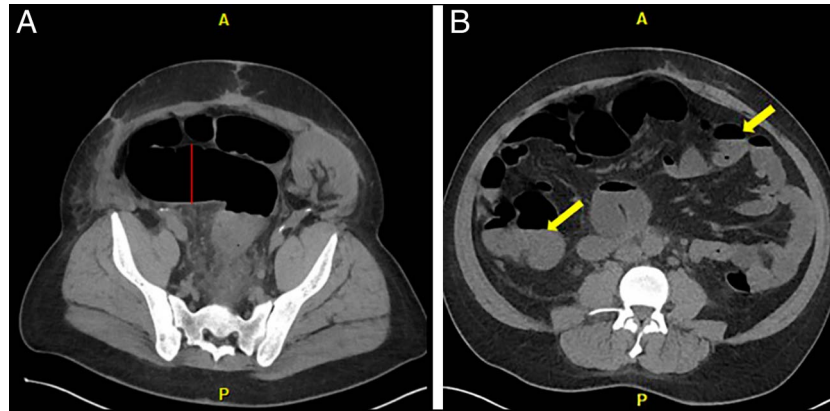


Figure 3. Evolving bowel obstruction on computed tomography. (A) New distention of the sigmoid colon up to 5.6 cm (red line) at the rectosigmoid junction. (B) Segments of small bowel dilatation (up to 5.0 cm) with air fluid levels (yellow arrows). No discrete transition points identified. A, anterior; P, posterior.

recommended conservative therapy with serial abdominal examinations because the patient was still able to produce flatus. A small-bowel follow-through was then completed; contrast was able to traverse to the colon after 2 hours, ruling out complete small bowel obstruction. By hospital day 5, the patient's symptoms had gradually improved to tolerate an oral diet. However, his eosinophilia had continued to rise daily to an absolute eosinophil count of $1.68 \times 10^9/L$ (12.5% eosinophils). For suspicion of adverse drug reaction, tecovirimat was discontinued. Additional causes of eosinophilia, including stool ova and parasite analysis, were negative. By hospital day 8, the patient's urinary and rectal symptoms had improved enough for Foley catheter removal and discharge home.

DISCUSSION

Mpox is a zoonotic orthopoxvirus infection that has affected more than 50 countries and is an evolving public health concern.⁵ The disease is generally self-limited, but severe cases can arise in immunocompromised patients. Gastrointestinal manifestations of mpox include nausea, vomiting, diarrhea,odynophagia, anorexia, rectal pain, and proctitis.^{1,5} Studies show that anorectal pain and proctitis is seen in 14% to 36% of patients.^{5,6} More severe symptoms such as rectal perforation, rectal abscess, or bowel obstruction are described in other case reports.^{1,7} Our case demonstrates how mpox proctitis can generate profound rectosigmoid inflammation to produce a bowel obstruction pattern in the small and large intestines. Urinary tract involvement can lead to progressive urinary retention. This case occurred during the Fall 2022 outbreak of mpox in the United States, where there was an incidence of 400 cases per day according to the Centers for Disease Control and Prevention. Healthcare providers should report cases of mpox to the local department of health to control the spread of the mpox outbreak.

Tecovirimat is a US Food and Drug Administration-approved antiviral medication for the treatment of smallpox with investigational expanded use for non-variola orthopoxvirus

infections, including mpox.⁸ Tecovirimat should be considered for severe disease or high-risk populations, such as immunocompromised, pregnant, and pediatric patients.³ In our case, there was suspicion of tecovirimat drug reaction causing eosinophilia. Newer studies suggest that severe mpox can cause elevated monocytes and granulocytes, such as eosinophils.⁹ This case supports severe mpox-induced eosinophilia because alternate causes were ruled out using history, physical examination, and stool studies to exclude parasitic infection.

In conclusion, we present a case of mpox severe proctitis complicated by bowel obstruction. Recognition of gastrointestinal manifestations of mpox can reduce delays in diagnosis and guide management.

DISCLOSURES

Author contributions: H. Saleh, BT Ho, JT Call, MM Plante, HB Zaver, HR Powers, AM Metcalfe, CG Graham, and MD Lewis contributed to multiple aspects of patient care and drafting/critical revision of this manuscript. All authors approve the final draft submitted. H. Saleh and MD Lewis are the author guarantors.

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