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From Crohn's Mimic to Rare Complication: A Case of Actinomyces Bacteremia in an Immunocompromised Patient with Crohn's Disease

Claire Harrington BS*, Esteban Figueroa and Brian Behm

University of Virginia School of Medicine 1215 Lee Street, Charlottesville, VA 22908, USA

*Corresponding author: Claire Harrington BS, University of Virginia School of Medicine, 1215 Lee Street, Charlottesville, Virginia 22908, USA, Tel: 757-2148-973; E-mail: claireharrington93@gmail.com

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Abstract

Abdominal actinomycosis has been described in the literature as a Crohn's disease mimic due to its indolent course and propensity to cause abscesses and fistulae. Actinomycosis rarely presents as an infectious complication in patients with IBD. We present a case of a 21-year-old male with Crohn's disease on immunosuppression, statuspost diverting loop ileostomy that developed a pelvic abscess complicated by Actinomycos bacteremia. Appropriate diagnosis of Actinomycosis can be delayed due to the fastidious growth in anaerobic blood cultures. Treatment involves a prolonged course (6-12 months) of antibiotics.

Keywords: Abdominal actinomycosis; Crohn's disease; *Actinomyces;* Immunosuppression

Introduction

Actinomyces is an anaerobic Gram-positive bacillus that is a normal component of the endogenous flora of the human oropharynx, gastrointestinal tract, and genitourinary tract [1,2]. Actinomyces infection classically presents as orocervicofacial actinomycosis, with thoracic and abdominal actinomycosis being less common presentations.

Abdominal actinomycosis can mimic inflammatory bowel disease (IBD) due to its indolent course and propensity to cause abscesses and fistulae [3-6]. Rarely, abdominal actinomycosis has been reported as an infectious complication in patients with IBD7-9, but monomicrobial *Actinomyces* bacteremia has not been previously described in the literature as a complication in a patient with IBD.

We present a case of a young man with Crohn's disease who developed a pelvic abscess complicated by *Actinomyces* bacteremia. Treatment of actinomycosis involves prolonged antibiotic therapy (6 to 12 months) with penicillin or amoxacillin-clavulanate [1].

Case Report

A 21-year-old man with Crohn's disease treated with infliximab and methotrexate who was status-post total proctocolectomy with ileoanal pouch anastomosis and diverting loop ileostomy presented to the emergency department with a chief complaint of lower abdominal pain, rectal bleeding and fever.

The patient was initially diagnosed with IBD two years prior after presenting with severe colitis at an outside institution. Colonoscopy at that time was consistent with pancolonic ulcerative colitis. Shortly after diagnosis, his course was complicated by *Clostridium difficile* infection and due to progressively worsening symptoms he underwent a 3-stage total colectomy with J pouch.

The year after takedown he began having problems with pouch dysfunction, failed several courses of antibiotics and eventually underwent diverting loop ileostomy in hopes salvaging his pouch. Despite the ileostomy he continued to be symptomatic and pouchoscopy one month prior to his presentation revealed perianal fissuring and active inflammation of the pouch with biopsies demonstrating chronic inflammation and granulomata of the pouch. The granulomatous inflammation was felt to be most consistent with Crohn's disease, and he was started on infliximab and methotrexate a month prior to his presentation.

On presentation to the emergency department (ED) the patient complained of progressively worsening rectal and abdominal pain for the past week and intermittent fevers. The abdominal pain started as right lower quadrant but had progressed to be diffuse in nature. In the ED, his vitals were significant for a blood pressure of 106/59 and a temperature of 101°F. Physical exam revealed abdominal tenderness in bilateral lower quadrants without guarding.

Laboratory work up was significant for a mild leukocytosis to 11.39 k/µL and his C-reactive protein (CRP) was elevated to 11.3 mg/dL. He had a normal comprehensive metabolic panel, and a normal lactic acid of 1.6 mmol/L. A CT scan of the abdomen and pelvis with contrast revealed worsened inflammatory stranding surrounding the J pouch when compared to his most recent imaging one month prior, and interval development of a new 2.4 cm by 3.3 cm rim-enhancing fluid and gas collection concerning for abscess adjacent to the J pouch (Figure 1).

This was not amenable to percutaneous drainage. The patient had blood cultures drawn and was treated with intravenous piperacillintazobactam empirically for the pelvic abscess. Stool testing for *Clostridium difficile* and community GI pathogens PCR panel were negative.

On hospital day 3, the patient demonstrated mild clinical improvement; however, his blood cultures from admission grew grampositive rods and eventually was speciated as *Actinomyces*. Infectious disease consultation was requested and given the patient's clinical presentation with pouch inflammation with pelvic abscess, it was

determined the *Actinomyces* bacteremia was likely a complication from abdominal Actinomycosis.

The patient was given a 2-week course of intravenous piperacillintazobactam followed by a transition to an oral course of amoxicillinclavulanate (875 mg-125 mg BID) for 6 months. Repeat blood cultures were negative and the patient was discharged home with a Peripherally Inserted Central Catheter (PICC) line to complete his course of antibiotics.

For management of the patient's Crohn's disease methotrexate and infliximab were held until interval imaging demonstrated resolution of his pelvic abscess. The patient was ultimately restarted on infliximab 5 mg/kg every 8 weeks and methotrexate 15 mg weekly.



Figure 1: CT abdomen pelvis demonstrating a $2.4 \text{ cm} \times 3.3 \text{ cm}$ fluid and gas collection (red arrow).

Discussion

Our patient's case provides an example of an atypical manifestation of a rare infectious complication in IBD. While there is a single case report describing polymicrobial *Actinomyces naeslundii* and *Pseudomonas aeruginosa* sepsis in a patient with ulcerative colitis [6-13], to our knowledge no case of monomicrobial Actinomyces bacteremia complicating IBD has previously been reported.

Actinomyces bacteremia is a rare clinical entity due to the fact that actinomycosis usually spreads contiguously, violating tissue planes rather than via hematogenous or lymphatic spread of infection [14]. It is thought that existing mucosal injury or inflammation is necessary in order for Actinomyces spp. to become pathogenic [3].

There is evidence that the dysbiosis of IBD results in higher population of Actinomyces genera compared to healthy controls [11]. The additive insults of ulceration of intestinal mucosa and abnormalities in epithelial tight junctions found in Crohn's disease may provide appropriate conditions for the development of actinomycosis. In addition, immunocompromised hosts are at increased risk for infection with Actinomyces [15-19].

Diagnosis of actinomycosis can be challenging. Isolation of Actinomyces spp. requires at least 5 days of anaerobic culture in enriched media, as was the case in our patient [20]. Actinomycosis can also be diagnosed histopathologically by visualization of sulfur granules on biopsy [9,10]. Pathology reviewed the patient's J-pouch

biopsies from one month prior to his admission, but no sulfur granules were appreciated on biopsies at that time.

Preferred treatment of actinomycosis is 6 to 12 months of penicillin; however, resistance to β -lactams is rare and amoxicillin-clavulanate is an acceptable regimen [1]. In patients with an allergy to penicillin, alternatives include doxycycline, ceftriaxone, clindamycin, and erythromycin [1].

In conclusion, this case illustrates the need to have high index of suspicion for infectious complications when taking care of patients with IBD, especially those treated with immunosuppression. Atypical clinical presentations should prompt early evaluation for atypical infections including actinomycosis, as diagnosis is frequently delayed or elusive due to the fastidious nature of Actinomyces.

Disclosure

The authors declare that there is no conflict of interests regarding the publication of this manuscript.

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Page 3 of 3

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