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Strongyloides Stercolais Hyperinfection in ANCA Vasculitis

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Abstract

Strongyloidiasis presents a spectrum of manifestations, ranging from asymptomatic infection to disseminated or potentially fatal hyperinfection. Immunosuppression, particularly induced by corticosteroids or HIV infection, is the most common trigger for life-threatening dissemination of larvae, resulting in mortality rates as high as 85%. We present a case of ANCA vasculitis that initially presented with skin rash, diarrhea, and abdominal pain. Initially evaluated for a potential flare of vasculitis, the patient was ultimately diagnosed with strongyloidiasis.

Keywords: strongyloidiasis; immunosuppression; dissemination; ANCA vasculitis

Introduction: Case presentation

A 54-year-old female with a history of GERD and p-ANCA vasculitis presented to the emergency department in context of dull aching epigastric pain and profuse watery diarrhea for 3 days. She described her abdominal pain as intermittent, crampy, non-radiating with no aggravating and relieving factors. She had 8-10 episodes of bowel movement per day with no blood or mucus in the stool and most of the times associated with abdominal pain. The patient's medical history was notable for fatigueness, nausea and several episodes of vomiting along with a itchy, purplish, slightly raised macular rash on her bilateral wrist, anterior abdomen and left lower extremity **(Figure 1)** that started within three days of her return to the US from Ethiopia about 1 month ago. At that time her hospital course was complicated by acute kidney injury with azotemia. She was found to have elevated inflammatory markers, p-ANCA, MPO ANCA. Given her proteinuria and hematuria, she underwent kidney biopsy that showed pauci-immune cresentric glomerulo nephritis consistent with ANCA vasculitis. She was treated with IV pulse steroid which was transitioned to oral steroid on discharge along with dapsone for PJP prophylaxis. Dermatology was consulted for rash who suggested differentials to be vasculitis versus paraneoplastic process versus ecchymosis in the setting of uremic platelet dysfunction. Dermatology recommended skin biopsy however patient opted for wait and watch. She was discharged three days prior to current presentation.

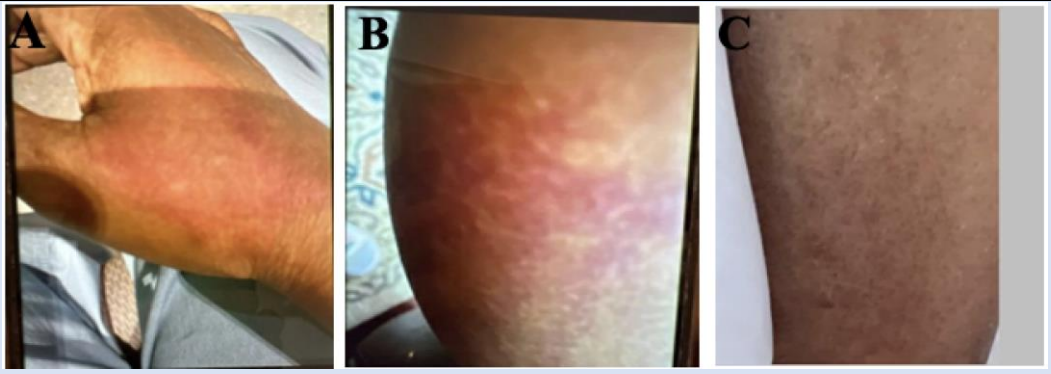

Figure 1: Demonstrates purplish, slightly raised macular rash on dorsum of right hand (A), anterior abdomen (B) and left thigh (C)

On presentation, the patient was afebrile, normotensive (110/70 mm Hg) with preserved respiratory rate and oxygen saturation. Examination was notable for bibasilar crackles on lungs. Laboratory tests revealed

normocytic anemia (hemoglobin 7.1 g/dl, reference range: 11.0-14.5 g/dl; MCV 84 fL, reference range: 81-100 fL), normal WBC and platelet counts, creatinine of 3.56 mg/dl (reference range:0.5- 0.8 mg/dL, baseline: 2.4 mg/dL), elevated lactate of 4 mmol/L (reference range 0.7 to 2 mmol/) L. PCR for clostridium difficile toxin was negative and stool ova and parasite smear was negative for white blood cells, ova or parasite.

The day after the admission, patient's hemoglobin dropped to 5.5 mg/dL. Her vitamin B12 level and folate level, LDH, ferritin, iron saturation was normal. Her serum urine immunoelectrophoresis were negative for any monoclonal spike. Her hemoglobin electrophoresis was normal with normal kappa and lambda ratio. She denied any hematemesis, melena or menorrhagia Chest x-ray showed pulmonary venous congestion. CT chest, abdomen and pelvis without IV contrast showed no significant abnormality or acute inflammatory process of gastrointestinal tract. She received 2 units of blood transfusion after which her hemoglobin improved to 7.7 gm/dL. She was started on maintenance fluid and Imodium as needed for loose stool. Her prednisone 25 mg for vasculitis was continued along with pantoprazole 40 mg twice daily. She was receiving morphine 2 mg every 4 hour as needed for abdominal pain.Four days after her hospitalization her diarrhea got resolved however patient had ongoing abdominal pain without any nausea or vomiting.

She underwent upper GI endoscopy that showed grade A reflux esophagitis with no bleeding, erythematous mucosa in the stomach **(Figure 2A)** which was biopsied, and duodenal erosion with stigmata of recent bleeding. She was started on sucralfate tablet 1gm per oral four times daily while waiting for pathology result. Her hospital course was further complicated by rapid response for hypotension with systolic blood pressure in the range of 80-90 mm Hg and altered mental status. Her repat hemoglobin level was 6.4 gm.dl with worsening creatinine of 3.7 mg/dl. The stomach antrum biopsy showed gastric mucosa with parasite infection consistent with strongyloides stercolais **(Figure 2B)** for which she was started on per oral ivermectin 12 mg once daily for seven days. Blood culture was sent and she was also started on piperacillin tazobactam for empiric gram-negative coverage. Her abdominal pain and rash were improved after starting ivermectin.

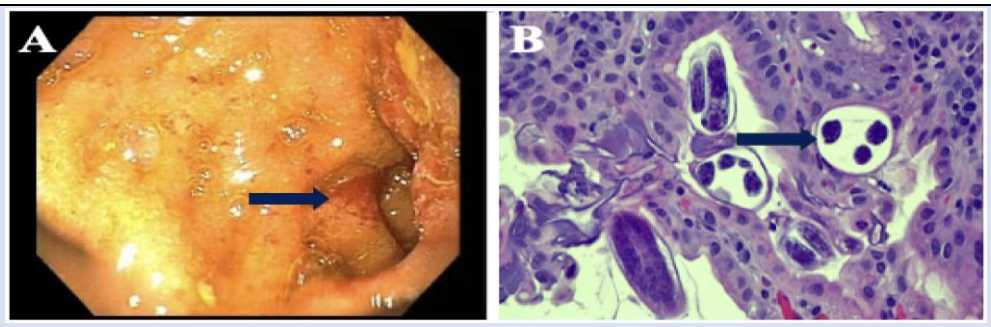

Figure 2: Demonstrates erythematous mucosa in the stomach antrum as evident in upper GI endoscopy (A) and stomach antrum biopsy showing strongyloides (B).

Discussion

Strongyloides stercoralis is a parasitic roundworm that inhabits the intestines and affects over 100 million people globally [1]. It is unique in its ability to exist as a free-living and autoinfective cycle [2]. Immunosuppression, especially from corticosteroid use or HIV infection, is the primary factor that can lead to the severe spread of larvae throughout the body, with mortality rates reaching as high as 85% [3]. Strongyloidiasis is primarily found in migrants and travelers returning from endemic regions in tropical and subtropical areas [1]. The association between systemic corticosteroid treatment and the spread of *S. stercoralis* has been more frequently reported [4]. *Strongyloides stercoralis* is unique among human-infecting nematodes because larvae expelled in feces can develop into a free-living generation of worms, which then produce infective larvae capable of penetrating the skin [3]. The parasite naturally infects dogs, primates, and humans, and is transmitted either through skin contact or the fecal-oral route [3]. It can present a wide range of symptoms, including cutaneous and gastrointestinal issues, but more than 60% of cases are asymptomatic, often only detected through an elevated eosinophil count in the blood [2]. Diagnosis can be challenging due to the low parasite load and irregular larval output [5]. A single stool

sample often fails to detect larvae in up to 70% of cases [6]. Eosinophilia is typically the only sign of *S. stercoralis* infection, but it is usually mild [5%–15%] and nonspecific [6]. The *Strongyloides* antibody test can cross-react with other helminth infections, such as filariasis, *Ascaris lumbricoides*, and acute schistosomiasis [6]. Biopsy of the duodenum or jejunum provides a diagnosis in about 90% of cases [7]. Patients with chronic strongyloidiasis who are immunosuppressed are at high risk of developing strongyloides hyperinfection syndrome, a potentially fatal condition where the rapid proliferation of larvae causes systemic sepsis and multiple organ failure. When diagnosed early, strongyloidiasis can be effectively treated with oral antihelmintic medications [8]. Treatment options include ivermectin, thiabendazole, and albendazole [5,9]. In one study by Datry et al, parasitological cure was achieved in 24 out of 29 patients treated with ivermectin (83%), compared to 9 out of 24 patients treated with albendazole (38%) showing Ivermectin to be more effective than albendazole [6].

Conclusion

Not all rashes are linked to autoimmune or specific dermatological conditions; they can also be caused by infections, which are often overlooked. Despite advancements in diagnostic techniques for detecting strongyloidiasis, particularly in complicated cases of hyperinfection or dissemination, there is still a lack of comprehensive guidelines for screening in epidemiological studies [2].

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