Rapid development of migratory, linear, and serpiginous lesions in association with immunosuppression

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CASE SUMMARY

History

A 78-year-old Bulgarian woman presented to the National Institutes of Health (NIH) with a diagnosis of poorly differentiated metastatic carcinoma of unknown origin. The prior month she had been seen at a hospital in Bulgaria for weight loss and a right inguinal mass. NIH pathology review confirmed a poorly differentiated carcinoma with extensive necrosis suggesting squamous cell carcinoma. She was enrolled in a treatment trial at NIH with metastatic disease invading the lungs and lymph nodes (mediastinum, abdomen, and pelvis) and a chemotherapy regimen was started of gemcitabine, carboplatin, and lenalidomide with dexamethasone as an antiemetic. The patient returned on day 8, and a rash of 2 days duration was noted. Immediately before arriving at the dermatology clinic, she developed altered mental status with aphasia and was admitted for neurologic observation. The altered mental status resolved and evaluation revealed only small-vessel ischemia. The patient was also experiencing diarrhea and was found to have elevated transaminases (4- to 7-fold over normal). Chemotherapy was held because of the transaminase abnormalities and altered mental status. The following day, the patient was seen by dermatology for a progressive asymptomatic eruption.

Physical examination

This elderly woman had multiple thin, urticarial, pink papules and plaques, ranging in size from 0.5 to 1.5 cm, and linear pink to red threadlike lesions arrayed over her trunk and extremities. Some lesions were arcuate. Across chest, breasts, the areolae, and thighs were striking, linear pink, palpable threads ranging from 2 to 6 cm in length (Fig 1). The total number of lesions was approximately 75, and none had epidermal change.

Histopathology

A punch biopsy specimen was obtained from a pink linear lesion on the right shoulder and showed very mild superficial perivascular lymphocytic infiltrate and numerous eosinophils in the dermis.

Significant diagnostic studies

Before the chemotherapy and systemic glucocorticoid treatment, the patient had an elevated white blood cell count of 10.14 K/μL (normal to 10.04 K/μL) and an elevated absolute eosinophil count of 0.81 K/μL (normal to 0.36 K/μL), but at the time of dermatology consultation her absolute eosinophil count was normal (0.22 K/μL). Aspartate aminotransferase was 144 U/L and alanine aminotransferase was 300 U/L. Microscopic evaluation of the stool revealed numerous rhabditiform larvae of *Strongyloides*

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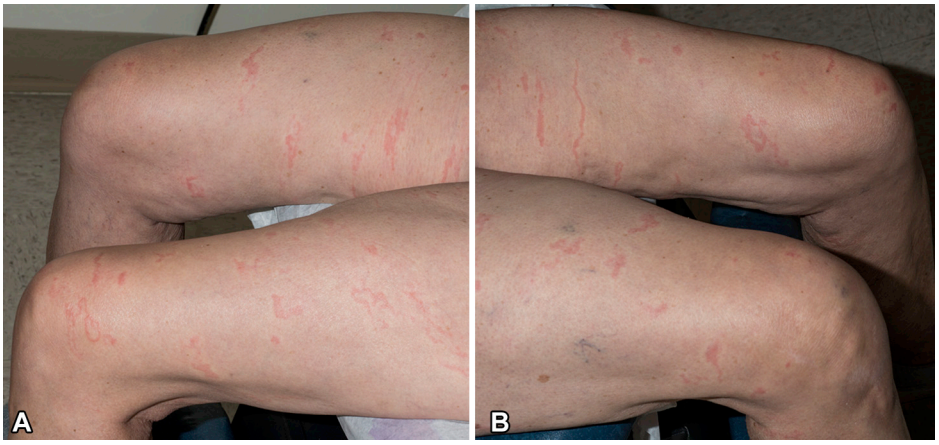

Fig 1. **A** and **B**, Larva currens. Linear and serpiginous, migratory pink threadlike lesions.

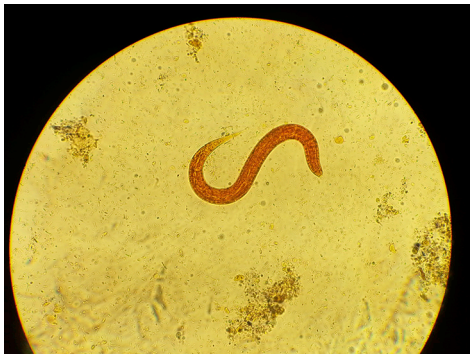

Fig 2. *Strongyloides stercoralis*. Microscopic evaluation of the stool revealed rhabditiform larvae.

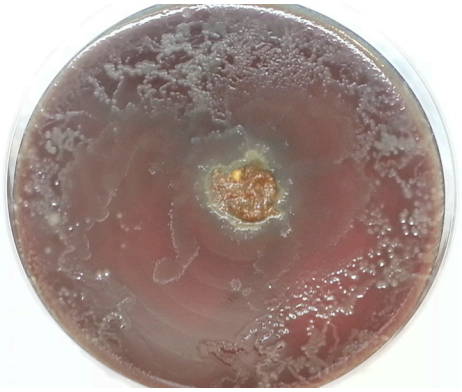

Fig 3. *Strongyloides stercoralis*. Stool inoculated in the center of a blood agar plate shows stool bacterial colonies in numerous serpiginous tracts, consistent with the presence of multiple live, motile larvae.

stercoralis (Fig 2). Stool inoculated in the center of a blood agar plate (Fig 3) demonstrated stool bacterial colonies in numerous serpiginous tracts, consistent with the presence of multiple live, motile larvae.

Diagnosis

The diagnosis of *Strongyloides stercoralis* hyperinfection syndrome was made.

FOLLOW-UP

The patient received a 7-day course of ivermectin. Within 4 days the rash resolved and the transaminase elevations improved. Repeated stool cultures were performed on days 4 and 7 of treatment, and both were negative for larvae. She resumed her chemotherapy and antiemetic dexamethasone after ivermectin treatment and confirmation of negative stool examinations for larvae.

DISCUSSION

Strongyloides stercoralis is a parasitic nematode (roundworm) with a worldwide distribution that includes the southeastern United States and southern Europe.[1] Infection is often asymptomatic in the absence of immunosuppression. Among nematodes, *Strongyloides stercoralis* is unusual in its ability to complete its entire life cycle in human beings through autoinfection and multiplication (Fig 4). Infection most commonly occurs through contact with infested soil. Free-living filariform larvae penetrate the skin, enter the circulatory system, and migrate through the lung before being coughed up and swallowed. In the small intestine, the larvae mature into adult worms. A female worm can produce up to 40 eggs per day. The eggs hatch into noninfective rhabditiform larvae, which are excreted in the stool. In a normal host, small numbers of rhabditiform larvae become filariform larvae in the large intestine. These filariform larvae can penetrate the perianal skin or migrate through the intestinal mucosa, reentering the circulatory system. This autoinfection cycle may persist for decades.

In immunosuppressed patients, parasite numbers can increase, accelerating the autoinfection cycle and causing clinical signs and symptoms termed the