blood count at this time revealed a platelet count of \(41 \times 10^3/\text{mm}^3\). Isotretinoin therapy was suspended and new treatment was instated with 100 mg/d prednisone; oral contraceptive medication was maintained. After 9 days, the platelet count had returned to normal \((179 \times 10^3/\text{mm}^3)\) and prednisone was suspended. The Coombs test and tests for antinuclear antibodies, anticardiolipin, human immunodeficiency virus, hepatitis B and hepatitis C virus, rheumatoid factor, antistreptolysin O, and antiplatelet antibodies were negative. The platelet count remained normal 18 months later.

Isotretinoin has been shown to cause a long list of secondary effects, including thrombocytopenia, of which only 4 cases have been previously reported.\(^1\)-\(^4\) The test for antiplatelet antibodies is usually positive in thrombocytopenia induced by isotretinoin.\(^6\) This test was negative in our patient, suggesting that the process was mediated by nonimmunologic mechanisms. We cannot rule out the implication of the oral contraceptives in this case, though we believe it to be improbable.

**References**


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**Giant Blister Due to Cutaneous Larva Migrans**

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**To the Editor:**

In recent years, the prevalence of exotic imported parasitic diseases has risen considerably within Europe due to tourism and migrational movements. Most of these diseases are characterized by cutaneous lesions; cutaneous larva migrans (CLM) is particularly common.\(^1\)

A 26-year-old man with no relevant history came urgently to our clinic due to a pruritic skin lesion on the sole of the right foot from 7 days previously, with no history of prior injury. He reported no general malaise or other systemic manifestations.

The physical examination revealed an inflamed, serpentine lesion with papules and vesicles, located on the inner side of the right foot (Figure 1), that rapidly progressed to form a giant blister that hindered walking (Figure 2).

Additional tests included a complete laboratory workup and chest radiograph that showed no significant abnormalities. The clinical diagnosis was CLM and treatment was started with albendazole 200 mg every 12 hours for 5 days. The patient recovered completely and the lesions gradually disappeared over 10 days.

CLM is a parasitosis caused by penetration and migration of nematode larvae through the skin. At present, these larvae are usually acquired in tropical regions with warm, humid climates, and the most important etiologic agent is *Ancylostoma braziliense*, although *A caninum* and *Uncinaria stenocephala*\(^2\)-\(^3\) are other species implicated. Humans are an inappropriate host for these parasites and, therefore, only experience cutaneous lesions (the larva remains in the skin without completing its life cycle, as it is unable to cross the basement membrane due to a lack of the necessary enzymes\(^4\)).

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**Figure 1.** Serpentine lesion with papules and vesicles located on the inner side of the right foot.

**Figure 2.** Giant blister on the right foot.
Clinically, the initial lesion is a papule that appears a few hours after penetration. The typical lesions appear within 4 to 6 days and are characterized by slightly elevated, mobile and migratory, sinuous and erythematous paths, 2 to 4 mm wide and 15 to 20 cm long, with a vesicle of serous content at the terminal end. Rare manifestations, such as folliculitis, hive-like rashes, and tinea pedis, have been reported. As the larva migrates, it travels between several millimeters and up to 1 or 2 cm per day, particularly at night, tracing a readily recognizable path. The lesions can be found on any part of the body exposed to the source of contamination (predominantly soles, back, buttocks, knees, and thighs) and cause severe pruritus, which leads to insomnia and scratching that can result in secondary infections. The skin rash may be accompanied by eosinophilia, elevated immunoglobulin (Ig) E levels, and even pulmonary infiltrates with eosinophilia, caused by hematogenous dissemination of the larva (Loeffler syndrome).

The diagnosis is mainly based on clinical symptoms, particularly the obvious serpentine lesions and difficulty to isolate the parasite in a skin biopsy. Several authors propose the use of epiluminescence microscopy to confirm the diagnosis and enzyme-linked immunosorbert assay techniques to detect specific IgG.

Several papers report the use of thiabendazole, elevated immunoglobulin (Ig) E levels, and even pulmonary infiltrates with eosinophilia, caused by hematogenous dissemination of the larva (Loeffler syndrome).

The larva usually disappears by itself within 1 to 6 months; however, our patient experienced severe pruritus and extreme discomfort that required adequate treatment. Although several therapeutic options, such as cryotherapy, topical thiabendazole, and oral thiabendazole (50 mg/d) are available, the current treatment of choice is albendazole (400–800 mg/d for 5 days). Flubendazole (200 mg/d for 5 days) and ivermectin (200 µg/kg as a single dose) may be therapeutic options in the future.

The patient we describe had a large blister caused by CLM of unknown pathogenesis, although we believe that its formation may be due to 1 of 3 mechanisms: 1) delayed hypersensitivity reaction, with the resulting release of unknown antigens from the larval infection; 2) irritation or contact allergic reaction caused by the topical treatments (our patient had not used any previous treatment), or 3) release of lytic enzymes by the larva itself.

Given the excellent therapeutic response with albendazole and the absence of adverse reactions, we consider, along with other authors, that this drug should be considered the first-choice treatment.

References