sign of paronychia and no involvement of other fingernails or toenails.

Fungal cultures of the nail plate showed the presence of *Fusarium* species. Routine blood tests including full blood count, erythrocyte sedimentation rate, and biochemistry gave completely normal results, and the patient tested seronegative for HIV.

Treatment with itraconazole (200 mg/d over 4 weeks) was prescribed, and a clear clinical improvement was seen.

Fusarium species are nondermatophyte molds that tend to occur naturally as saprophytes in the soil and as pathogens in plants.[1] Although infections can occur in humans, they are normally precipitated by local or general predisposing factors. Systemic infections are rare and only occur in immunodepressed patients.[2] Onychomycosis caused by *Fusarium* species almost always affects the great toe, especially when accompanied by dystrophic and traumatic abnormalities or where nails were previously infected with dermatophytes.[3] Wearing sandals or walking barefoot can encourage the condition.[2] Most of the cases described in the literature are characterized by extensive paronychia, but this was not the case in our patient.

Invasion of the toenail by *Fusarium oxysporum* is relatively uncommon despite the widespread distribution of the mold, implying that the toenail may be a portal of entry for systemic infections in immunodepressed patients,[4-6] with the associated worse prognosis.

The condition manifests itself clinically as the typical form of proximal subungual onychomycosis, occasionally with the presence of onycholysis or subungual hyperkeratosis.

Treatment of *Fusarium* onychomycosis is not a straightforward matter.[2] Better levels of response are reported with ungual avulsion followed by the application of topical antifungal agents, giving an improvement in most immunocompetent patients. Itraconazole (200 mg/d over 4 weeks or in pulse therapy) has also been used with some success.[2]

We would like to present the case of an immunocompetent patient who was diagnosed with proximal white subungual onychomycosis caused by *Fusarium* species.

We also stress the value of microbiological cultures and the need to rule out immunosuppression—especially by HIV infection—in such ungual disorders, which are almost exclusive to immunodepressed patients and rarely diagnosed in immunocompetent individuals.

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Temporary Thrombocytopenia Probably Induced by Isotretinoin

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To the Editor:

Isotretinoin is a drug that is widely used to treat severe nodular or cystic acne.[1] It can cause serious adverse effects that should be recognized and monitored by clinicians. We report a case of profound thrombocytopenia due to treatment of severe acne with isotretinoin. This case illustrates a serious adverse effect that can occur at any stage of treatment. A review of the literature revealed only 4 studies on this topic.[2-5]

A 29-year-old Caucasian woman with nodular and cystic acne refractory to other therapies began treatment with 40 mg/d isotretinoin after providing written informed consent. The patient was taking no other medication except oral contraceptives (ethinylestradiol and cyproterone acetate), which she had begun 3 years earlier. The contraceptive medication was maintained. All laboratory test results prior to treatment (including biochemistry and blood counts) were normal.

A month later, the acne had improved significantly and treatment with isotretinoin was well tolerated, except for cheilitis. Further biochemistry and blood counts were normal. No other medication was prescribed during this period.

Six months after beginning treatment, the patient visited our department due to spontaneous vaginal bleeding that had begun 10 days earlier and was not related to menstruation. A petechial exanthema was visible on the torso and limbs. A

blood count at this time revealed a platelet count of 41 × 10^3/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 × 10^3/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.

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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).

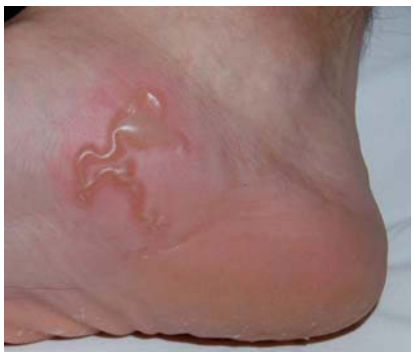

Figure 1. Serpentine lesion with papules and vesicles located on the inner side of the right foot.

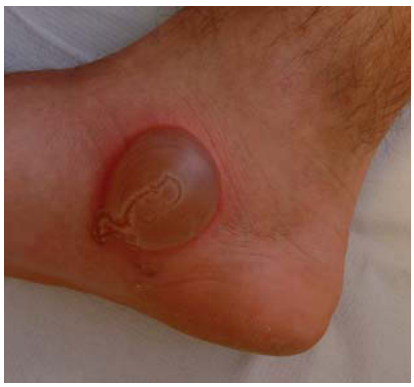

Figure 2. Giant blister on the right foot.

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]).