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Case Report

Foot mycosis caused by fusarium solani mimicking tinea pedis in pediatric patient

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Abstract Dermatophytes, yeasts, and non-dermatophytic molds (NDMs) are the most common causal agents of foot mycoses. It is uncommon in women and children. *Fusarium* species are widely distributed and can be detected in several environmental sources such as soil, air, and plants. Some predisposition factors could be related with this disease including barefoot, wearing occlusive shoes, sweating of the feet. The most common cause of localized *fusarium* infection is trauma. A 11-year-old boy, complained of itching and red patches on right and left feet over the last year. Patient used to play barefoot, usually on the ground, in the fields and often played when it was raining. Patient also had a history of being stabbed by a twig. Patient used to wear socks for a long time at school. On physical examination at right and left plantar pedis regions showed multiple erythematous macules with firm margins,covered with thin scales on top, plaques with yellowish crusts on top, erosions,no pus and no pustules. Potassium hydroxide 20% examination revealed fungal hyphae, and white and cotton macroscopic fungi were found from fungal culture examination. Pink or violet center, and becoming bluish-brown are present sporoconidia. Microscopically, it showed sickle or canoe shaped thin-walled macroconidia with 3–4 septa, microconidia, chlamydospore. Showed concordance with the feature of spesies namely *fusarium solani.* Patients received itraconazole 100 mg twice a day for 1 month and cetirizine 10 mg once a day, clinical cure was noted and mycological cure was obtained after 1 month of treatment. Foot mycosis due to non-dermatophyte species, namely *fusarium solani* is a rare case. Clinical manifestation might mimick dermatophyte foot mycosis. A detail history and physical examination, supported by identification of etiology will lead to correct diagnosis and prompt treatment. Education of patient on how to prevent reccurency by avoiding predisposition factor of the disease is the most important point of care.

Key words
Foot mycosis; Fusarium solani; Tinea pedis.

Introduction

Foot mycoses are generally caused by dermatophytes, sometimes yeasts, and seldom and in unusual situations by non-dermatophyte molds (NDMs). To date, it is uncommon in women and children. The mechanisms of human infection are not fully understood. The duration of incubation is unknown. Several transmission mechanisms have been proposed, including inhalation, implantation, trauma, and acquisition via contaminated intravascular devices. Trauma is the most common cause of localized *Fusarium* infection in immunocompetent people.[1] In humans, *Fusarium* species can potentially produce a disease that is localized, focally invasive or disseminated. Infection in otherwise healthy people is unusual, yet the pathogen still causes illness.

Infections produced by fungi belonging to the genus *Fusarium*, which can spread to plants, animals, and humans, are known as fusariosis.[2]

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Fusarium species are widespread plant diseases found in organic substrates, water, and soil.[3] The majority of soil microbes, called saprobes, are completely innocuous and can be found in great numbers.[4] There are over 50 described species of *Fusarium*, with the *F solani* and *F oxysporum* species' complexes being responsible for 70% of invasive human diseases.[3] *Fusarium* spp. are distinguished by well-developed, septated, non-pigmented hyphae with acute-angled bifurcations that form macroconidia, also known as sporodochial conidia.[5] *F. verticillioides, F. solani, F. proliferatum, F. oxysporum, F. nivale, F. moniliforme,* and *F. chlamydosporum* have all been linked to human illness.[1] The *Fusarium solani* species combination and the *Fusarium oxysporum* species complex were shown in a recent American study to be the third and fourth most prevalent causes of IFI in children.[3] In this report, we wanted to draw attention to foot mycosis caused by *Fusarium solani* mimicking tinea pedis.

Case report

A patient came to our institution with the chief complaint of itchy red patches on the soles of the feet. These complaints of red patches appearing on the soles of the right and left feet began one year prior to arrival. Initially, a thickened patch appeared on the sole of the right foot that felt itchy, causing the patient to scratche continuously, resulting in a pus-filled blister, which burst and left scars and crusts. Currently, the spots on the soles of the feet were sore. The patches on the palms of the hands initially appeared filled with pus and eventually broke leaving patches and scales accompanied by itching. Any history of red patches on the body was denied. The patient has a history of often playing in the rain in the fields, barefoot. The patient has a history of being pricked by thorns/ twigs.

The patient was not from a boarding school. He reported rarely wear sandals when at home or play in outside home. Complaints of smelly feet were denied. History of taking drugs other than those prescribed by the doctor, drinking herbal medicine or herbal medicine, smearing with oil and history of wearing shoes over a long period of time, or history wearing socks for a long time were denied. History of diabetes mellitus, having allergy of specific food, eggs, chocolate, milk, fish and chicken were denied. History of asthma and sneezing in the morning were denied. Patient's mother and father both had food allergies. The history of treatment from a dermatologist at Petrokimia Gresik Hospital included ketoconazole, desoximetasone, carmed, griseofulvin, cetirizine, hydrocortisone but the existing complaints had not improved. On physical examination, the weight and height of the patient were 57 kg and 150 cm respectively. The patient's blood pressure was 110/80 mmHg, heart rate 80 x/min, respiratory rate 20 x/min

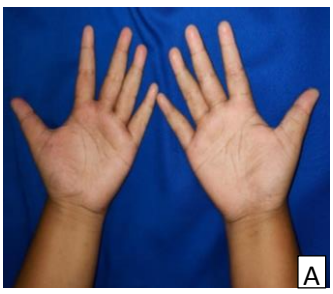

Figure 1 Before treatment, (A) Region manus dextra et sinistra, no abnormalities were found. (B) Multiple erythematous macules with firm margins with thin scales on top, plaques with yellowish crusts on top, erosions seen, no pus found, no pustules.

Figure 2 Progression of the lesions after one month of treatment. (A) Regio Manus Dextra et Sinistra, no abnormalities were found. (B) Not found erythematous macules, no pus, no vesicle bullae, minimal scaly.

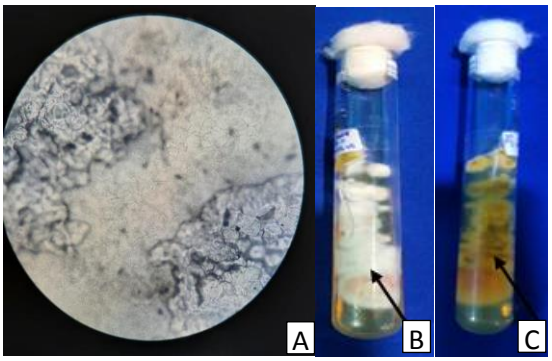

Figure 3 (A) KOH 20% was found hyphae. (B,C) Culture result showed white and cotton. Pink or violet center, and becoming bluish-brown are present sporoconidia.

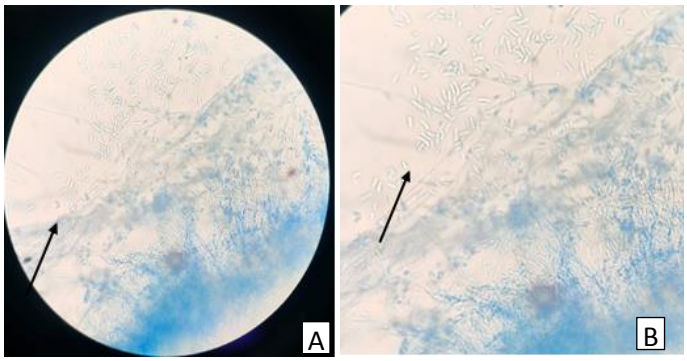

Figure 4 (A) Magnification 200x, the colony microscopically showed sickle or canoe shaped thin-walled macroconidia with 3-4 septa, microconidia, chlamydospore suitable with the features of Fusarium solani. (B) Magnification 400x.

and body temperature was 36.4˚C. Patient's BMI was 25 (over body weight). The glasgow coma scale of patient was E4V5M6. There were no abnormalities found on general status examination. Local status examination at right plantar pedis showed multiple erythematous macules with firm margins and thin scales on top, plaques with yellowish crusts on top, erosions, no pus found and no pustules. Local status at left plantar pedis sinistra showed multiple erythematous macules, well-defined margins with thin scales on top, erosions, no pus found and no pustules. No nail abnormalities were found at right and lft dorsal pedis. The differential diagnosis were tinea pedis, phompolix/ dyshidrotic eczema and scabies. Further examinations were then carried out.

Culture fungi result after 1 weeks showed white and cottony. Micelium white to cream and becoming bluish-brown. Microscopic examination with 400x magnificient showed sickle-shaped thin-walled macroconidia with 3–4 septas, microconidia, chlamydospor. This finding suggested *Fusarium solani*. The patient was then assessed as foot mycosis caused by *Fusarium solani*.

Discussion

Fusarium spp. Is known to cause several diseases such as interdigital foot infections, onychomycosis, keratitis, and others.[4,6,7] Most cases of *Fusarium* sp-related integumentary lesions appeared as erythematous, nodular papules with core necrosis.[2] Clinical patterns that suggest an inflammatory response persist even though the lesions are typically confined and appear as keratitis and onychomycosis in immunocompetent patients.[1] In immunocompromised patients, *Fusarium* spp. have been linked to significant fatality rates and widespread infectious diseases.[1]

From an epidemiological vantage point, interdigital intertrigo caused by *Fusarium* spp. is extremely uncommon and it causes sores between the toes.[6] *Fusarium onychomycosis* is responsible for a range of 0.97% to 6% of onychomycosis cases, with *Fusarium* sp being identified as the causal agent in 9 to 44% of non-dermatophyte mold (NDM) onychomycosis cases.[8] Several cases of tinea pedis caused by *Fusarium solani* have been published.[9]

Fusarium species produce a wide range of diseases in humans, from superficial and local infection sites to disseminated infections, with onychomycosis, keratitis, and skin infections being the most common.[2] Patients with hematological tumors, organ transplant recipients, and burn victims were previously known to be at increased risk for developing disseminated fusariosis.[4]

Severe hyperkeratosis, characterized by bilateral, well-demarcated erythema with minute papules on periphery, fine white scaling, and hyperkeratosis restricted to heels, soles, and lateral border of foot, has also been attributed to *Fusarium* sp. In this case, the clinical manifestation included the appereance of bilateral, erythematous macules, white scales, and hyperkeratosis in soles.

Serology, blood culture, mycology, gram stain, and histopathology can all help diagnose *Fusarium* infection.[10] Local skin infections are easily identified by physician inspection. *Fusarium* sp. can grow quickly on a variety of typical nutrient agars, such as potato dextrose agar or Sabouraud agar. *F. solani* grows at a little slower rate than *F. oxysporum.*[5]

Clinical manifestations suggest that *Fusarium* sp causes a severe hyperkarototic, bilateral, well-demarcated erythema on the heels, soles, and lateral border of the foot with minute papules on the periphery, fine white scaling, and hyperkeratosis. In this case, the clinical manifestation appearence were bilateral, erythematous macules, white scaling, and hyperkeratosis in soles. At right and left plantar pedis regions, there were multiple erythematous macules, well-defined margins with thin scales on top, hyperkeratotic, with no erosions, pus found or pustules.

Mycosis caused by nondermatophytic molds can be diagnosed by looking for fungal elements in 20% KOH-preparations obtained from scrapings of the nail and skin. Three separate cultures from the same patient at weekly intervals all showed identical mold growth. These cultures showed no evidence of dermatophytes or yeasts.[11] Based on KOH examination, the result usually revealed as large, distorted, septate, nondermatophytic hyphae. In this case, the KOH examination taken from regio pedis shows hyphae. Regarding fungi culture test, the result usually shows a white and cotton appereance fo colony morphology. The result also shows sickle or canoe shape. In this case, the result of fungi culture test showed sickle-shaped thin-walled macroconidia with 3-4 septas, microconidia, chlamydospore which indicate as *Fusarium solani.*

Patients with hematologic malignancy or bone marrow transplant who may experience prolonged or severe neutropenia during treatment should have their skin and nails thoroughly examined, as these areas are common sites of infection and can serve as entry points for the spread of infection throughout the body.[10] Since cutaneous lesions can be noticed at an early stage of the disease and in roughly seventy-five percent of patients of disseminated *Fusarium* sp infection, the skin can be a crucial and early hint to diagnosis.[10] The high mortality rate is caused by invasive Fusarium sp infections, which promote an insufficient host response to the infection.[2] The respiratory system, the digestive system, and the skin are all potential entry points for a virus that has the potential to spread throughout the body.[10]

Fusariosis can be considered with several differential diagnoses such as tinea pedis, scabies, and dyshidortic eczema. Patients with tinea pedis mostly present with symptoms of intertriginous dermatitis that are long-lasting, itchy, and characterized by maceration, peeling, and cracking. In tinea pedis, chronic, scaly thickened plaques usually appear on the plantar surface of the foot which are usually referred to as “moccasin slippers”. The clinical manifestations of fusarium infection can resemble tinea pedis if the port of entry for the infection is in the pedis area (if there is a wound on the foot). The differential diagnosis can exclude tinea pedis based on microscopic examination with the presence of *Fusarium sp.* hyphae. However, there is still the possibility of co-occurrence between these two diseases.

The clinical appearance of fusarium and scabies may occur in similar locations. As the predilection for scabies is most often in the axilla, around the umbilicus, genitals, buttocks, volar part of the wrist, between the fingers, elbow flexors, and the palms and soles of the feet. To rule out the differential diagnosis of scabies, it can be confirmed that there is no history of night itching and coexistence in densely populated communities. Microscopic examination should not reveal any mites in fusariosis.

Dyshidrotic eczema is a condition associated with atopy. Exogenous factors such as contact dermatitis to balsam, nickel, cobalt, dermatophyte infections or bacterial infections are known to trigger this episode. Many patients report recurrence of this eczema during periods of stress. This reaction pattern is supported by the fact that it is well known that this eczema clears up when the tinea pedis infection is treated and returns when the fungal infection returns. In dyshidrotic eczema, the palms and sides of the fingers develop distinct vesicles and/or bullae. The soles of the feet and the outside edges of the toes may also be harmed. Vesicles are usually limited to the lateral sides of the fingers and to the foot region only in mildly affected patients. It usually heals without rupture, followed by desquamation. Examination of potassium hydroxide wet preparation to eliminate dermatophyte infection.

As is known, *Fusarium sp.* infection usually occurs in patients with an immunocompromised status whose infection can trigger severe clinical symptoms, but in this patient, with an immunocompetent status, the symptoms appear to be localized. there was no history indicating immunocompromised status in this patient.

The treatment of foot mycosis has become increasingly difficult in recent years, with many patients reporting treatment failure and recurrence. Fusariosis can be prevented and treated with amphotericin B, voriconazole, and posaconazole; these drugs have not changed much over time.[2] Posaconazole has been demonstrated to be 50% effective in curing fusarium infections in patients with neutropenia.[1] In this case, the patient was given first-line treatment with Ketoconazole 2x200mg and then treated with itraconazole 2x200mg for 14 days. *Fusarium* spp. are known to be intrinsically resistant to echinocandins, and some isolates are also resistant to azoles. Amphotericin B and voriconazole are the medications of choice in this setting, and drug combinations are an important approach of combating multi-drug resistance.[2] Amphotericin B in high dosage, typically in the liposomal formulation, as well as voriconazole and posaconazole, have both been shown to be effective in treating disseminated illness.[10]

Conclusion

Mycosis of the feet due to non-dermatophyte species, namely *Fusarium solani*, is a rare case, especially in the pediatric group. Clinical manifestation might mimick dermatophyte foot mycosis. The patient's immune status also plays an important role where in immunocompetent patients, the symptoms are often milder than in immunocompromised conditions. A detailed history and physical examination, supported by identification of etiology will lead to correct diagnosis and prompt treatment. In this case, the administration of the itraconazole regimen showed signs of improvement in the patient's clinical status. Education of the patient on how to prevent reccurency by avoiding predisposition factors of the disease is the most important point of care.

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