Case Report

Tinea Corporis in a Patient with Non- Hodgkin Malignant Lymphoma with Peripheral T Cells

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ABSTRACT

Background: Tinea corporis is a superficial infection of the skin caused by Dermatophytes. It is characterized by inflammatory or non-inflammatory lesions. It is also called the ringworm of the body. Clinically, it represents with rash that becomes ring-shaped, with itchy, scaly border and a clear center.

Case report: The patient E. C was diagnosed seven months ago at the hematology service with "non Hodgkin Malignant lymphoma with peripheral T cells. The patient, on his third round of treatment, represents with disseminated erythematous elements in the skin, ring-shaped rash spread throughout the body; in the neck, nose, face, legs and arms. He complained of itching and pain. Further laboratory examination of skin for fungi was requested. The microscopic examination of the skin with KOH resulted positive for septate hyphae. Trichophyton rubrum was isolated in the culture. The patient was treated during the hospital stay with oral Terbinafine 250 mg/day and Ketoconazole creams 2%. After therapy there were no signs of tinea corporis but only cutaneous elements of lymphoma.

Conclusion: We presented a case of an immune-compromised patient with extensive Tinea corporis caused by T. rubrum. Early diagnoses and proper treatment are important in the progression of the disease.

Keywords: Dermatophytes, non Hodking Malignant lymphoma, septate hyphae, tinea.

INTRODUCTION

Tinea corporis is a superficial infection of the skin caused by Dermatophytes and characterized by inflammatory or non-inflammatory lesions. It is also called ringworm of the body. Clinically, it shows a rash that becomes ring-shaped, red colored with a scaly border and a clear center.¹,² Dermatophytes are likely to live in the corneal layer of the skin, because of its warm and moisturizing features favorable for the dermatophytes development. They produce enzymes that invade the skin and generally do not penetrate deeply due to non-specific defense mechanisms.

CASE REPORT

Patient, E. C born in 1972, resident in Vlora was hospitalized at the Hematology Service, University Hospital Center on June 17, 2013. The patient was diagnosed with "non Hodgkin’s Malignant lymphoma with peripheral T cells. The patient presented...
with general pruritus, weakness, fever, dexter cervical adenopathy with diameter 4 cm and peripheral edema in the joints. The hemograma showed: WBC 81000/mm³, RBC 405000/mm³, Hb 11.1gr/dl, PLT 80000/mm³. Abdominal ultrasound revealed a liver of 18cm, lien 21cm, both without focal lesions. A grouping of limfonodules was seen in the hepatic hilus with dimensions 43 x 22 mm. Paraortic limfonodules were seen as well. Biopsy of sinister axilar limfonodul showed a structure partially destroyed by lymphatic infiltration representing irregular nuclei with average dimensions. Cells have invaded and destroyed some of the lymphoid follicles. Follicles were small and irregular. An increase in plasma cells and eosinophilic leukocytes in residual sinususes was observed. The limfonodul capsule and the perinodal adipose tissue were infiltrated by irregular lymphocytes. Immune-phenotyping showed irregular medium sized CD45 (+), CD3 (+), CD5 (+) lymphocytes. The positivity of CD20 and CD23 was limited to residual follicles. BCL2 was positive in a part of irregular lymphocytes. Ki-67 index was about 40-45% in diffuse areas. BCL6 and Cycline D1 did not give any specific result. The patient comes for treatment (third cycle) and represents with ring shaped erythematous elements in the skin spread throughout the body; in the neck, nose, face and extremities (as shown in figure 1-a and 1-b). He complained of itching and pain.

The patient has not been in contact with animals. From the hematology service was requested examination of skin for fungi. Laboratory examination: A KOH test was used to visualize fungal elements in the corneal layer of the skin. The sample was taken from the active border, because this part offers the highest concentration of fungal elements. Fungal culture was performed in Sabouraud dextrose agar monitoring the incubation temperature in 28 C for 4 weeks. (3)

RESULTS

The microscopic examination of the skin with KOH resulted positive for septate hyphae (as shown in figure 2).

T. rubrum was identified based on colony morphology on Sabouraud's dextrose agar (as shown in figure 3). Microscopic examination of material obtained from culture revealed pyriforme microconidia (as shown in figure 4-a and 4-b).
Urease and hair perforation test were performed for Trychophyton species identification. (3)

**DISCUSSION**

Tinea corporis can be manifested in different ways. Typically, the lesion begins as a scaly, scratchy, ring-shaped, erytematous rash that may deteriorate rapidly. (4,5) Immune-compromised patients often have atypical presentation of the disease. It may appear as a disseminated infection of the skin or as deep abscesses with severe itching or pain. (6,7)

The exact mechanism of cutaneous element forming is not known. It is believed that fungal virulence and immune response of the organism play a role in its pathogenesis. T. rubrum is the most common species of anthropile dermatophytes. The ability to produce proteolytic enzymes is a major virulence factor. T. rubrum invades human skin through destruction of keratin. Keratin is a fibrous protein and an important structural component of human skin. Infections can be transmitted from one person to another through direct contact. (8)

Although not life threatening, infections are long lasting, repetitive and difficult to cure. T. rubrum was isolated in immune-
compromised patients from other authors. \(^9\) The patient was treated with Terbinafine 250 mg / day for 2-4 weeks and ketoconazole creams 2%, 2 times a day. \(^10,11\) After therapy there were no signs of tinea corporis but only cutaneous elements of lymphoma.

CONCLUSION

We presented a case of an immunocompromised patient with extensive Tinea corporis caused by T. rubrum. Early diagnoses and proper treatment are important in the progression of the disease.

REFERENCES


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