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Norwegian Scabies in Diabetic Patient : A Case Report and Literature Review

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Abstrak

Norwegian scabies, also called crusted scabies is a parasitic disease of the skin, caused by the mite Sarcoptes Scabiei (var. hominis), from the classic form of the disease is characterized by papules, blood crusts, pustules, small vesicles and tunnels also called furrows. In Norway in 1848, discovered a variety of scabies with a peculiar clinic in patients with lepromatous leprosy that they later named Norwegian, crusty or hyperkeratotic scabies. It occurs in patients with immune disorders such as patients with leprosy, diabetics, patients with Down syndrome, mental disorders, in patients treated with glucocorticoids and in patients with HIV / AIDS. The early diagnosis of this disease is complex, due to its low frequency and the atypicality of its presentation, however, the diagnosis can be made by identification tests for the mite or its eggs, such as direct examination with 10% potassium hydroxide (KOH) of the scales that are shed from the affected skin lesions. Oral ivermectin and 5% topical permethrin have been considered the treatment of choice according to past guidelines for the management of this variety of scabies. Regarding the topical treatment, permethrin 5%, benzyl benzoate 25%, can also be used in classic scabies precipitated sulphur. In this particular case, a medical treatment was chosen with oral ivermectin doses of 200 µg / kg with repeat doses and 1% topically, benzyl benzoate was not chosen due to the effect of local irritation and dermatitis.

Keywords*: *Sarcoptes scabiei, Norwegian Scabies, Diabetes Mellitus, Immunocompromised

INTRODUCTION

Norwegian scabies, also called crusted scabies is a parasitic disease of the skin, caused by the mite Sarcoptes Scabiei (var. hominis), from the classic form of the disease is characterized by papules, blood crusts, pustules, small vesicles and tunnels also called furrows. The itching present in this pathology is very characteristic and is more frequent at night. The female parasite is the one that maintains the parasitosis, she travels 2.5 cm on the surface of the skin and advances from 2 to 3 mm per day in the tunnel that digs in the corneal layer. The male dies after copulation and the female lays 2 to 4 eggs daily that pass through the larval, nymph and adult stages in fourteen days.[1]

In Norway in 1848, Danielssen and Boeck discovered a variety of scabies with a peculiar clinic in patients with lepromatous leprosy that they later named Norwegian, crusty or hyperkeratotic scabies. It occurs in patients with immune disorders such as patients with leprosy, diabetics, patients with Down syndrome, mental disorders, in patients treated with glucocorticoids and in patients with HIV / AIDS. Norwegian scabies begins to manifest as the classic form of scabies, there may be a decrease in itching that sometimes makes diagnosis difficult. The grooves that represent the elemental lesion of the disease may not be visible or may become hyperkeratotic and the skin may acquire an erythematous appearance with psoriatic plaques with

thick scales of 3 to 15 mm of yellowish colour that, when detached, leave the appearance of crusted skin.[2,3]

The early diagnosis of this disease is complex, due to its low frequency and the atypicality of its presentation, however, the diagnosis can be made by identification tests for the mite or its eggs, such as direct examination with 10% potassium hydroxide (KOH) of the scales that are shed from the affected skin lesions. Skin biopsy can be requested in cases of diagnostic doubt or poor response to treatment, it is diagnostic when parasites are found in any stage of their biological cycle or characteristic alterations in the epidermis (acanthosis, hyperkeratosis, thickened corneal layer with multiple tunnels or burrows), where the inflammatory infiltrate is nonspecific. Treatment is difficult due to the large number of parasites that exist in the skin scales and crusty lesions of the patient.[4,5]

CASE REPORT

We present a clinical case of a 40-year-old male patient who presented to the dermatology service's outpatient clinic with a disseminated dermatosis involving the trunk, lumbar region, gluteal region, genitalia, thighs, axillary folds, interdigital spaces, and back of feet. It is polymorphous in appearance and is characterized by multiple papules that are extremely itchy, hyperkeratotic, erythematous, squamous plaques with thick whitish adherent scales and hematic crusts as a result of scratching. In the first instance, he was treated with corticosteroids, antihistamines, and ciprofloxacin as a symptomatic measure and was diagnosed with nonspecific dermatitis. He did not improve clinically. A mycological examination was requested, which confirmed no fungi. There was no direct examination of the injury. Patient did not attend his check-ups on a regular basis. Patients have insufficient control of type 2 diabetes mellitus despite prior use of metformin (850 mg / day) and glibenclamide (5 mg / day) and arterial hypertension under treatment with amlodipine (10 mg / day) and enalapril (10 mg). /day). Laboratory data indicate that HbA1c is 9.7%, fasting blood glucose is 320 mg / dL.

When the condition did not improve and the patient's son and wife began exhibiting similar symptoms, an incisional tissue sample of a lesion on the abdomen skin was requested for greater diagnostic certainty. The results revealed a superficial and deep infiltrate of lymphocytes, histiocytes, mast cells, and eosinophils, as well as the presence of spongiotic vesicles in the epidermis, highlighting the presence of a burrow of a mite compatible with Sarcoptes Scabiei.

Ivermectin started with 200 µg / day and given on 1st, 2nd, 7th days and with a booster dose at 14th day, associated with topical treatment with 1% ivermectin indicated at night. The treatment was associated with hydroxyzine 25 mg / 8 hrs, general measures were recommended for both the patient and the family regarding the cleaning and repeated washing of clothing and bedding of the patient and the family group, which was given treatment with single dose ivermectin 200 µg. After seven days, control was conducted and the patient demonstrated significant clinical improvement of the lesions, although hyperchromic areas in the trunk region and desquamation in certain areas were still witnessed.

Figure 1. (1A) Hyperkeratotic lesion on the right (1A) and left (1B) auricular region, improved hyperkeratotic lession on both ears (1C, 1D)

Figure 2. Plaque hyperkeratotic lesion on the back region (2A) and thick plaque with scales on the gluteal region (2B), improved plaque hyperkeratotic lession on the back and gluteal region (2C, 2D)

Figure 3. Yellowish thick scales found on both of the palmar region (3A), improved yellowish thick scales (3B)

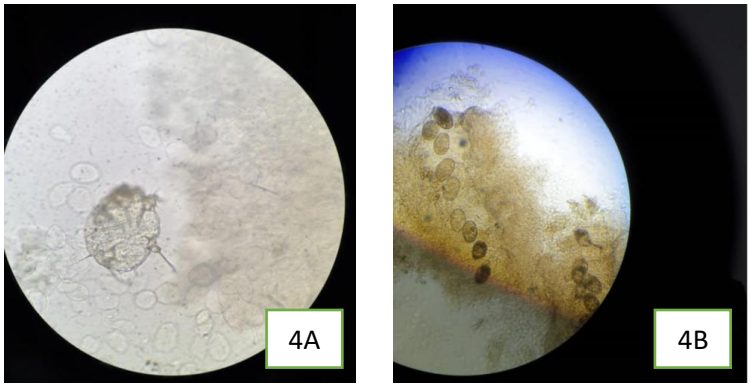

Figure 4. Skin scraping: Adult mite (4A), eggs (4B)

DISCUSSION

Norwegian scabies, also called crusted scabies is a parasitic disease of the skin, caused by the mite Sarcoptes scabiei (var. hominis), and usually found in immunocompromised patients. In the case presented, the patient has a history of poorly controlled Arterial Hypertension and Type 2 Diabetes Mellitus, which corresponds to a predisposing factor to suffer this clinical form of presentation, due to the compromise of adaptive cellular immunity causing hyper infestation.[6–8]

Unlike usual scabies, crusty scabies can affect any part of the body as we saw reflected in the patient. The morphology of these lesions, on physical examination, coincide with those of this variety of scabies and the evolution can be seen from a classic or classic form with papules, crusted lesions and “furrows” to the atypical form with hyperchromic, keratotic lesions that gave off thick scales highly contagious by the presence of mites. Although itching is not usually present in this clinical form, in this case we see it as an early and generalized symptom. Due to the atypical form of presentation and the low frequency, it is difficult to diagnose this entity only with the clinic, and it is sometimes overlooked. Eczema, psoriasis, Darier's disease, pityriasis rosacea, tinea versicolor, pediculosis of the body, ichthyosis, and lichen planus are cited as possible differential diagnoses. Complementary studies were used to demonstrate the presence of the parasite; Biopsy and histopathological study were useful in the diagnosis of this case, but other diagnostic methods already mentioned could have been used in the same way.[9,10]

Oral ivermectin and 5% topical permethrin have been considered the treatment of choice according to past guidelines for the management of this variety of scabies. Currently, these recommendations have not changed much, and alternatives are suggested with ivermectin in doses of 200 µg / kg twice a week apart between the doses. In severe cases of the disease, it is recommended to give oral ivermectin 200 µg / kg on day 1, 2 and day 8. If the clinical picture is very severe with persistence of the mites on the patient's skin, an additional dose of ivermectin may be required on days 9 and 15 or even on days 9, 15, 22 and 29, it has been seen that up to 92.5% of patients are cured with two or three doses of this drug.[2,11–13]

Regarding the topical treatment, permethrin 5%, benzyl benzoate 25%, can also be used in classic scabies precipitated sulphur. In this particular case, a medical treatment was chosen with oral ivermectin doses of 200 µg / kg with repeat doses and 1% topically, benzyl benzoate was not chosen due to the effect of local irritation and dermatitis, that could exacerbate the clinical picture of the patient. The pruritus was controlled with antihistamines until the treatment is effective. Due to the frequent association of lesions with over-aggregated skin and soft tissue infections, systemic antibiotics can also be used in some case.

Prophylactic treatment for close contacts of the patient has been recommended even when they do not present symptoms and consists of the administration of ivermectin 200 µg / kg in a single oral dose, as was done in this case.[14,15]

Due to the underlying disease of the patient, consultation with the endocrinology service was carried out as soon as possible, due to the counterproductive effect of poor management of diabetes in terms of the effectiveness of the treatment of crusted scabies. In the case presented, we are faced with a patient with an important risk factor that predisposes him to present this severe form of the disease. Diabetes, as we know, needs adequate glycaemic control to avoid complications of the same disease and thus also avoid opportunistic infections. To avoid complications in diabetic patients with poor glycaemic control and crusty scabies, we recommend early diagnosis and installation of adequate and effective therapeutic measures in the patient and close contacts.

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