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Case Report: Vesicular Toxicodermia Induced by the Scabies Mite Sarcoptes Scabiei

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Abstract

This article analyzes a clinical case of vesicular toxicodermia caused by infection with the scabies mite Sarcoptes scabiei. The focus is on a female patient who, according to her medical history, noticed the development of intense itching and rashes on her hands and feet after using an antiseptic. Initial treatment provided no relief, and further investigations confirmed the presence of the scabies mite. The article emphasizes the importance of a comprehensive approach to diagnosing and treating such clinical cases, including careful monitoring for allergic reactions to antiseptics and other potential irritants. Special attention is given to the necessity of etiological therapy directly targeting the scabies pathogen, as well as symptomatic treatment to alleviate itching and inflammatory manifestations.

Keywords: Vesicular toxicodermia, scabies mite Sarcoptes scabiei, scabies complication.

Introduction

Toxic-allergic dermatitis, or toxicodermia, is an acute inflammation of the skin caused by exposure to allergenic or toxic-allergenic factors, which can enter the body parenterally, through food, or in the form of vapors and aerosols [1, 2]. These factors can include a wide range of substances such as drugs, chemicals, and even biological agents. When these substances enter the body, they can trigger an immune response that manifests as skin inflammation. The severity of the reaction can vary, ranging from mild rashes to severe skin damage. Furthermore, the pathogenesis of toxicodermia often involves both immediate and delayed hypersensitivity reactions, contributing to its complex clinical presentation.

The French dermatologist J. Jadassohn first proposed the term «toxicodermia» in 1896. This condition most commonly occurs as a side effect of medications [3]. Although pathological reactions can develop to any drug, including antihistamines and glucocorticosteroids, toxicodermia is most frequently associated with the use of antibacterial agents, antiepileptic drugs, and allopurinol. The development of such rashes is linked to the predominance of CD4 and CD8 T-lymphocytes and delayed-type hypersensitivity [4]. This clinical case presents an example of toxicodermia as a complication of atypical scabies.

Case presentation

Patient T. presented with complaints of itching and rashes on the palms, shins, and feet. Disease history: the complaints began on October 26, when she noticed the appearance of isolated vesicular rashes on the skin of her palms, feet, and shins, which then spread further. She associated this condition with the use of an antiseptic at work. Over the past two weeks, she has noted the appearance of blisters. She was consulted

by an allergist at her place of residence and was prescribed Prednisolone 90-60-60 mg. She noted temporary improvement with the corticosteroid treatment, but the skin manifestations progressed over time. She received Metronidazole, Cefazolin, and Azithromycin for three days. Upon a repeat consultation with the allergist, Azithromycin was replaced with Ceftriaxone (antibiotic therapy was initiated based on signs of inflammation in the complete blood count). Despite the ongoing comprehensive therapy, her condition did not improve. She noted the bursting of vesicles, persistent skin itching, with increased intensity at night, making it difficult to sleep.

Allergic history: In 2014, she had a similar episode of rashes during her first pregnancy at 19-20 weeks, for which she received inpatient treatment with Prednisolone and was discharged with improvement. Occupational history: She works as a doctor, daily sanitizes her hands with antiseptic, and wears latex gloves.

Local Status: On the skin of the palms, feet, and shins, there are vesicles and papules with a diameter of 2-3 mm, oozing, linear scratch marks, and crusts (Figure 1).

Complete Blood Count (23.11.2022): Hemoglobin (HGB) – 138 g/L; Erythrocytes (RBC) – 5.110^12/L; Color Index – 0.81; Hematocrit (HCT) – 40%; Platelets (PLT) – 323.010^9/L; Leukocytes (WBC) – 7.1*10^9/L; Segmental Neutrophils (NEUT) – 73%; Basophils (BASO) – 6%; Lymphocytes (LYMPH) – 21%; Erythrocyte Sedimentation Rate (ESR) – 11

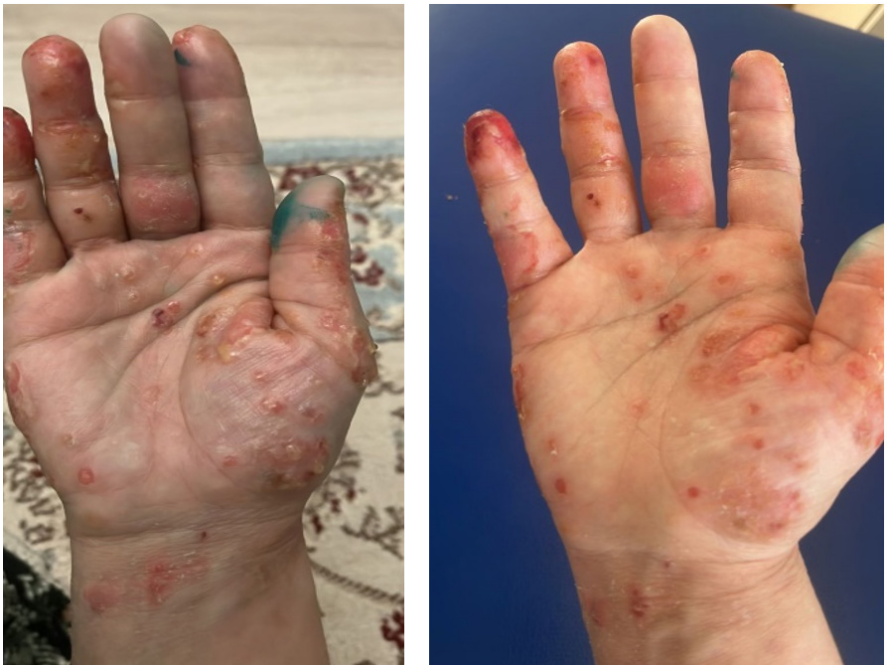

Figure 1 – Clinical Condition of the Patient on the Day of Presentation

Figure 2a – Dynamics of the Patient's Clinical Condition During Treatment – Patient's Condition on Day 5 of Treatment

Figure 2b – Dynamics of the Patient's Clinical Condition During Treatment – Patient's Condition on Day 5 of Treatment

Figure 2c – Dynamics of the Patient's Clinical Condition During Treatment – Patient's Condition on Day 7 of Treatment

mm/h. Biochemical profile (23.11.2022): Total Protein – 80; Alanine Aminotransferase – 16.9; Aspartate Aminotransferase – 20.0; Total Bilirubin – 10.9; Direct Bilirubin – 3.4; Glucose – 6.8; Urea – 5.6; Creatinine – 62. Coagulation Test (23.11.2022): Activated Partial Thromboplastin Time – 34.0 sec; International Normalized Ratio – 1.07; Prothrombin Index – 93%; Prothrombin Time – 15.0 sec; Thrombin Time – 13.4 sec.

On 30.11.2022, the patient visited the Regional Allergology Center "DiVera," where she was diagnosed with mild vesicular toxicodermia. The prescribed treatment included a hypoallergenic diet and the following medications: Prednisolone 90 mg (GCS) with isotonic NaCl solution 100 ml, administered intravenously once a day for 5 days; Cetirizine 10 mg (H1-histamine receptor blocker), 1 tablet once a day for 10 days; topically, Methylene Blue 3 times a day for 7 days. The patient was referred for further tests, including total IgE and specific IgE to latex, and a skin scraping for the detection of the scabies mite (Figure 2).

From the 5th day of therapy, the treatment regimen was changed to Prednisolone 5 mg, 1 tablet once a day in the morning; Cetirizine 10 mg, 1 tablet once a day; and Methyluracil cream applied topically twice a day.

Skin scraping for the detection of the scabies mite was positive. The diagnosis was established as atypical scabies without burrows, complicated by mild vesicular toxicodermia.

The patient was referred to a dermatologist at the dermatovenereology dispensary for further consultation.

Discussion

This article presents an example of toxic-allergic dermatitis against the background of scabies. The clinical case describes the severity of the disease, the dynamics of clinical symptom development, and the complexity of differential diagnosis and treatment.

Human scabies is a contagious skin disease caused by the parasitic mite Sarcoptes scabiei var. hominis. It is a common skin condition that remains a serious public health concern worldwide [5]. The clinical pathological signs of scabies are generally divided into two categories, depending on the type of hypersensitivity reaction: immediate immune response type I, mediated by antibodies [5-7], or delayed cell-mediated immune response type IV [8-10]. Type I reactions are characterized by immunoglobulin (Ig) E-mediated activation of mast cells and eosinophils, whereas type IV reactions involve sensitized T-cells that either cause direct damage or activate other leukocytes [11].

A meta-analysis by Næsborg-Nielsen in 2022 showed that the immediate immune response associated with type I hypersensitivity reaction in the context of scabies is primarily driven by a combination of IgE, mast cells, and eosinophils, followed by the release of histamine and other pro-inflammatory cytokines. The immune response associated with type IV hypersensitivity more frequently involved the proliferation of macrophages, neutrophils, and B-cells [12].

The study by Abd El-Aal in 2016 demonstrated elevated levels of total IgE in scabies [13], and the Australian study by Walton in 2010 found that patients with scabies had higher levels of specific IgE to recombinant scabies antigens compared to the control group [14]. Ito's 2011 study confirmed the presence of mast cells and basophils in the skin of scabies patients [15]. Upon activation, mast cells and basophils rapidly produce TNF-α, IL-6, Th2 cytokines IL-4, IL-5, and IL-13, which are key molecules responsible for Th2-type allergic inflammation [16].

Abd El-Aal's 2016 study also demonstrated the involvement of cytokines IL-10, IL-6, INF-γ и TNF-α in the immune response to scabies [13]. These cytokines play a crucial role in activating macrophages, which were found in small quantities in the skin of scabies patients. It was hypothesized that in the early stages of infestation, mites suppress the ability of macrophages to migrate to the site of inflammation, allowing the mites to grow and establish themselves [17]. Alongside macrophages, neutrophils are an essential part of the innate immune system in the development of type IV hypersensitivity. Luo's 2016 study presented histological data from skin lesions in 44 cases of bullous scabies, showing that neutrophils were the predominant inflammatory cell infiltrates [18]. In another similar study, 25 skin biopsies from scabies patients showed the presence of dermal neutrophils in 52% of cases [19].

At the XI Scientific and Practical Conference of Dermatovenereologists and Cosmetologists, a clinical case from 2016 to 2017 at the 4th department of the GBUZ "GorKVD" was described, where 15 patients with a diagnosis of scabies were treated. From the disease history, it was found that all patients had been treated at district dermatology and venereology clinics with diagnoses of allergic dermatitis and toxicodermia, receiving therapy without effect. Against the background of antiallergic therapy, the skin process progressed. Skin scrapings revealed a large number of scabies mites in all patients. Because an incorrect diagnosis was initially made, the treatment of the patients took longer than necessary. After detoxification, desensitization, and antibacterial therapy, all patients showed improvement, and the clinical symptoms of the disease decreased [20].

Conclusions

A thorough analysis of the medical history is a key element in diagnosing toxicodermia, involving the identification of similar symptoms in the patient's history, occupational risk factors related to the disease development, and the use of medications, among other aspects. In the clinical case under consideration, difficulties arose in identifying causal relationships with the etiological agent due to atypical clinical manifestations. The discussed example highlights that a comprehensive methodology in choosing diagnostic approaches is necessary for successful differential diagnosis and determination of the clinical diagnosis.

After referral to the dermatologist at the dermatovenereology dispensary, the patient was prescribed specific treatment for scabies, including Permethrin cream applied topically to all affected areas of the skin and oral Ivermectin. Additionally, symptomatic treatment with antihistamines and continued topical corticosteroids was recommended. Follow-up after two weeks of this treatment regimen showed significant improvement in the patient's condition: the vesicular and papular rashes had largely resolved, itching was substantially reduced, and there were no new lesions. This case underscores the importance of considering underlying parasitic infections such as scabies in the differential diagnosis of toxicodermia, particularly in patients with atypical presentations.

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