Pityriasis rosea in a patient with retrovirus infection and a history of syphilis and positive results of infection with hepatitis A virus, hepatitis B virus and hepatitis C virus

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Skin changes in the course of human immunodeficiency virus (HIV) infection appear to be a common phenomenon. According to different sources, such changes present in more than 90% of patients with the retroviral infection [1, 2]. Often these skin lesions are the first sign of immunosuppression and contribute to the onset of diagnostic tests in the direction of immune disorders [1–3]. It is believed that the dermatological symptoms in the course of HIV infection may be primarily related to infection with the virus or a secondary result of immunosuppression in the course of the disease [2]. An important issue is the occurrence of drug-induced skin changes associated with antiretroviral therapy and the treatment of diseases associated with HIV-positive patients, which can present in various forms.

A 34-year-old male patient presented to the dermatological emergency room due to scattered lesions, the first onset of which occurred about 4 weeks before. On interview, the patient reported a previous viral infection of the upper respiratory tract shortly before the onset of rash, and contact with animals (dogs, cats). In addition, he was infected with HIV (Stage A3 – according to the CDC AIDS classification, diagnosed in 2003), hepatitis B virus (HBV), hepatitis C virus (HCV) and had an illness history of syphilis (2009) and hepatitis A virus (HAV) infection. Furthermore, the examination of the patient's condition revealed hypertension, smoking, varicose veins of the lower extremities. The patient remains under control of the Acquired Immunodeficiency Clinic.

On admission, the patient's general condition was stable and without fever. On the skin's surface of the front and back of the trunk and upper and lower limbs, there were numerous eruptions of well-demarcated erythematous plaques of the circular or elliptical shape, with a gentle scaling on the surface and mild pruritus. (Figure 1).

Deviations observed in laboratory tests include: elevated liver enzymes, increased red blood cell volume and slightly elevated IgE antibody titer. Serological studies found: positive anti-HAV IgG, positive anti-HCV, anti-HBc and anti-HBs, and positive syphilitic reactions (TPHA, FTA-ABS). RNA viral load of HIV was below the cut-off point, while the number of CD4+ lymphocytes was 42 cells/mm^3 and CD8+ was 733 cells/mm^3.

Mycological examinations of scrapings taken from the lesions (direct preparation and culture were negative) and Wood's lamp examination (negative) were also conducted. Biopsies of skin lesions for histopathological examination concluded the image of superficial dermatitis without clear-specific features, which could correspond to pityriasis rosea or eczema (Figure 2).

The patient was treated both locally (glucocorticoids, antifungal preparations) and systemically (antibiotics, glucocorticoid, antifungals, antihistamines, calcium), however, failed to present a satisfactory dermatologic improvement. Approximately 8 weeks after the onset of skin lesions, the patient reported for a control visit, during which his general state was complete remission of the clinical disease (Figure 3). Taking into account the general and dermatological condition of the patient, results of additional tests, lack of response to treatment, and the spontaneous regression of lesions after a few weeks from the initial manifestation of symptoms, ultimately the diagnosis of pityriasis rosea Gibert was established.

Pityriasis rosea (PR) is a dermatosis from a group of erythematous-squamous diseases with an acute or subacute course, with an unclear etiology, and was first recognized in 1798 by Robert Will. The current name comes from the French doctor, C.M. Gibert, who first described the disease in 1860 [4].

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Prevalence of PR and clinical picture varies depending on the geographic region of the world, which is related to climatic and environmental conditions in different regions, as well as culture and health. Variance can be anywhere between 0.5% and 6% [5]. There was no significant difference in the incidence between men and women, but most sources argue a slight advantage in women [6], while children and young adults (75% of those affected in the range 10–35 y.o.) [7], pregnant women [5], immunocompromised patients [8] are groups shown to be particularly predisposed to the disease. There is also a family tendency of developing PR [6, 7]. In conclusion, among HIV-infected patients with atypical skin lesions and due to their increased predisposition for PR, differential diagnosis should expand the possibility of such a diagnosis.

The typical clinical course of PR is a prior infection (mostly of the upper respiratory tract in 69% of cases) [9], seasonal diseases (especially in spring and fall months), spontaneous recovery and immunological phenomena responsible by a viral etiology, and the most likely etiopathogenic factor is a virus from the HHV6 or HHV7 herpes family [10].

According to some authors, the course of skin lesions in PR is a systemic response to a HHV6 or HHV7 infection [11]. There have been reports of relapses (up to 3% of cases) [6, 7].

Due to the possibility of inducing dermatoses of the HHV6 virus, one study examined the impact of PR on pregnancy. Observed cases were that of preterm labor with neonatal hypotonia or miscarriage, especially when the infection occurred within the first 15 weeks of pregnancy [11].

The classical clinical picture of a patient with PR will present with an initial, single lesion, called "herald patch" (well demarcated, erythematous oval outbreak of salmon pink color, accompanied by peripheral scaling, gradually expanding to the size of 2–10 cm in diameter, usually located on the skin of the trunk or neck), which is present in approx. 80% of cases [12] where over a period of a few to a dozen or so days is the only change. This is followed by the seeding of numerous secondary eruptions (the next 2 weeks) of the skin, characterized by erythematous plaques, contracting symmetrically along the long axis parallel to dermatomes, which is mainly located on the trunk. In addition, lesions occur on the skin around the shoulder girdle, pelvis and proximal parts of the limbs. The face, hands and feet are usually not affected. No eruptions are observed on the mucous membranes [10]. Primary itching is not a characteristic sign. Typically, the disease duration is 6–8 weeks, but there are cases of shorter and longer duration [13]. In HIV-infected patients, the course of the disease is usually longer.

In addition to the classic form of PR, unusual clinical pictures differing in morphology, number, distribution of eruptions and the course and severity of symptoms are often found.

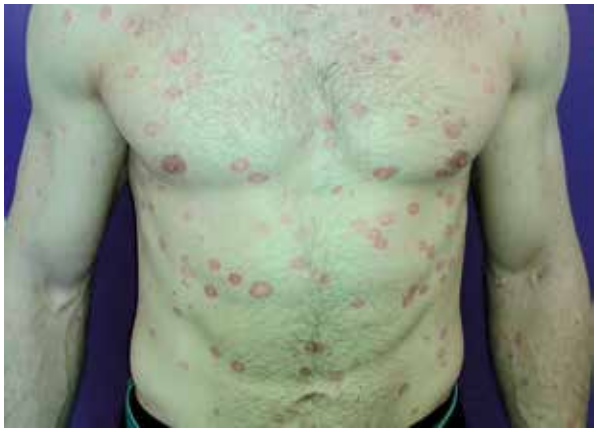

Figure 1. Skin changes on the day of admission

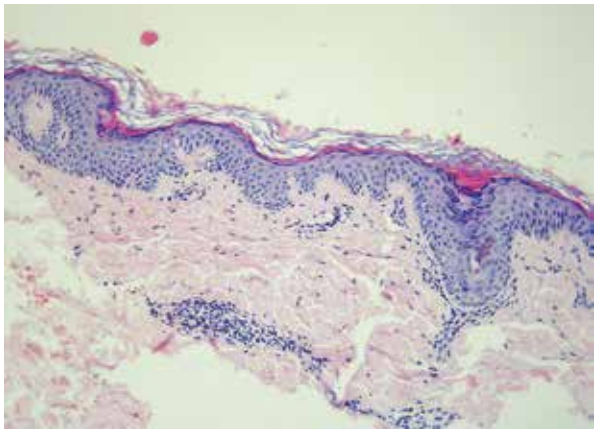

Figure 2. Skin biopsy: thin epidermis with features of moderate edema, single inflammatory cell infiltrates, with thickened, loose stratum corneum, without parakeratosis, and markedly clear granular layer; in the upper part of the epidermis, individual dyskeratotic cells and cytoid bodies; in upper layers of the dermis small perivascular inflammatory infiltrates and superficial hemosiderin deposits

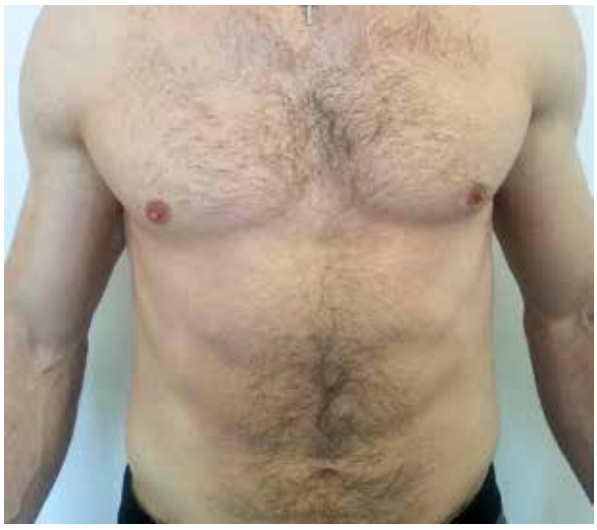

Figure 3. Complete remission 8 weeks after the onset of skin lesions

In one study examining a population of dark-skinned American children with PR, 30% of cases had localized lesions on the skin of the face, and 8% on the scalp [13]. A good example of the unusual course of the disease is the lack of a parent plaque which proved to be the case presented by us.

Histopathology, the case of PR is not characteristic and really allows one to exclude other diagnoses. It fulfills the criteria of superficial dermatitis [14].

In the differential diagnosis of PR, the following diseases should be considered: secondary syphilis (especially in HIV infection), seborrheic dermatitis, tinea cutis glabrae, tinea versicolor, nummular eczema, urticaria, erythema multiforme, pityriasis lichenoides chronica and different chemicals that may mimic the changes observed in PR (e.g. barbiturates, ACE inhibitors, gold salts, isotretinoin, terbinafine) [15].

Due to the self-limited course of the disease, treatment is not necessary. However, in the case of accompanying pruritus, application of topical corticosteroids and oral antihistamines is recommended. There are several studies on the efficacy and usefulness of treating PR, which prove the beneficial effects of macrolide antibiotics, especially erythromycin (at a dose of 250 mg four times a day, which significantly shortened the persistence of skin lesions) [9] and UVB phototherapy (reducing the overall severity of the disease) [16]. In another study [17], it was observed that treatment with acyclovir at a dose of 5 × 800 mg daily for 1 week accelerated the process of improvement. Considering the possible adverse effects of therapy, currently no form of treatment is recommended, perhaps due to the fact that spontaneous recovery occurs in 100% of patients. In the present case, due to the significant disturbances of the patient's immune system and the high risk of complications, it was decided to include multidirectional local or systemic treatment until there was a definitive diagnosis.

To sum up, pityriasis rosea is an often-occurring disease entity, with unclear etiology. Therefore, further studies are needed to examine and explain the reason for this dermatosis, such as determining the impact on the immune system and reactivation of HHV6 and HHV7 viruses, by inducing reactions in the form of skin lesions. As can be seen in the case described above, immune disorders may affect both the morphology and course of the skin disease.

Conflict of interest

The authors declare no conflict of interest.

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