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Janssen-Sponsored Satellite Symposium at the 30th EADV Virtual Congress 2021

The art of joint forces: crafting psoriatic arthritis care for dermatologists

This virtual satellite symposium will focus on the necessity for practicing dermatologists to understand the burden of psoriatic arthritis in patients with psoriasis. It will emphasize how important it is that dermatologists detect early signals of psoriatic arthritis in patients with psoriasis and also understand why targeting IL-23 directly can be effective in treating and potentially also preventing the development of psoriatic arthritis for their psoriasis patients

Janssen Immunology

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The differential diagnoses include tuberculosis, leishmaniasis, syphilis, bronchogenic carcinoma and other systemic mycoses. It has been suggested that co-infection with HIV and *P. brasiliensis* gives a mixed form of acute and chronic disease which represents a reactivation of a quiescent focus.[3]

The diagnosis is confirmed by identification of the typical budding yeast structures in tissue on direct microscopy and by mycological culture when possible. The growth in modified Sabouraud medium can take some weeks. Other tests such as serology and intradermal reaction have a role in the monitoring of the disease during treatment and in epidemiological studies. Detection of antibodies in the serum or antigen in body fluids is also useful in diagnosing the infection. The antibody tests may be less helpful in the immunocompromised.[4]

Our patient had the pathognomonic findings of yeasts in lesional skin and bronchoalveolar lavage. His serology has been persistently positive since diagnosis despite clinical and mycological cure, with 6 years of follow-up.

The evidence comparing different treatments in the management of paracoccidioidomycosis is very limited.[5] The use of the imidazoles itraconazole and ketoconazole is widely reported. Itraconazole is currently the drug of choice for treating this infection.[6] Amphotericin B, trimethoprim–sulfamethoxazole and terbinafine are also used.[7] Our patient showed a rapid sustained improvement following the use of itraconazole but remains under regular follow-up as relapse occurs in up to 5% of cases.[1]

Skin and oral lesions show diffuse staining for the inflammatory cytokine tumour necrosis factor, the expression of which is substantially reduced by day 20 of treatment.[8]

The diagnosis of paracoccidioidomycosis must be considered in individuals presenting with respiratory and/or mucocutaneous symptoms and who originate or are returning travellers from areas where *P. brasiliensis* is endemic.

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Key words: *imported, itraconazole, paracoccidioidomycosis, skin*

Conflicts of interest: none declared.

Cutaneous neonatal lupus erythematosus in four siblings

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Sir, Neonatal lupus erythematosus (NLE) is a form of lupus erythematosus (LE) first described in 1954,[1] representing a model of passive autoimmune disease in which pathogenetic autoantibodies are transmitted from mother to foetus through the placenta. Maternal autoantibodies are generally anti-Ro/SSA or anti-La/SSB, less frequently anti-U1RNP. NLE is characterized by a combination of dermatological, cardiac, haematological and hepatic manifestations. Cutaneous lesions consist of erythematous papules and annular discoid or polycyclic plaques, with or without fine scales. They can occur on sun-exposed areas, often with a typical periorbital distribution. Skin lesions can occur either at birth or, more often, within a few weeks after birth. They are transient and disappear along with autoantibody levels in 6–9 months, sometimes leaving permanent marks. Cardiac involvement occurs mainly with conduction defects and congenital heart block (CHB). Haematological abnormalities (haemolytic anaemia, neutropenia and thrombocytopenia) and hepatic involvement (abnormalities of liver function tests) are often asymptomatic and transient. Mothers may be affected by systemic LE, Sjögren syndrome or other autoimmune diseases, or may be clinically asymptomatic at the time of birth. We report four siblings, born to a woman affected by serological LE, who presented only the cutaneous form of NLE.

A 21-year-old woman from Tunisia had a history of fetal loss during her first pregnancy in 1997. During a second pregnancy, she developed fever, asthenia, headache and a transient malar rash. The symptoms were not investigated further and resolved spontaneously in 10 months. The successive pregnancies were uneventful.

Male A, born in 1998, presented annular hypopigmented macules upon the head, trunk and limbs during the first

month of life. These were diagnosed as a mycosis and were not investigated further. Spontaneous resolution was observed in a few months.

Female B, born in 2001, and two female monozygotic twins (C and D), born in 2002, showed a skin eruption in the first 2 months of life that was characterized by inflammatory annular plaques, with hyperkeratotic borders and atrophic centres (Fig. 1a). Lesions were distributed on the scalp, face, trunk and limbs. There was no history of sun exposure. Skin biopsy showed vacuolar degeneration at the dermal–epidermal junction, scattered necrotic keratinocytes and a perivascular mononuclear infiltrate in the dermis (Fig. 1b). Lesions progressively disappeared over a period of 6 months. While resolution was complete in the first two children, skin atrophy and hypopigmentation were present in twins C and D after 1 year.

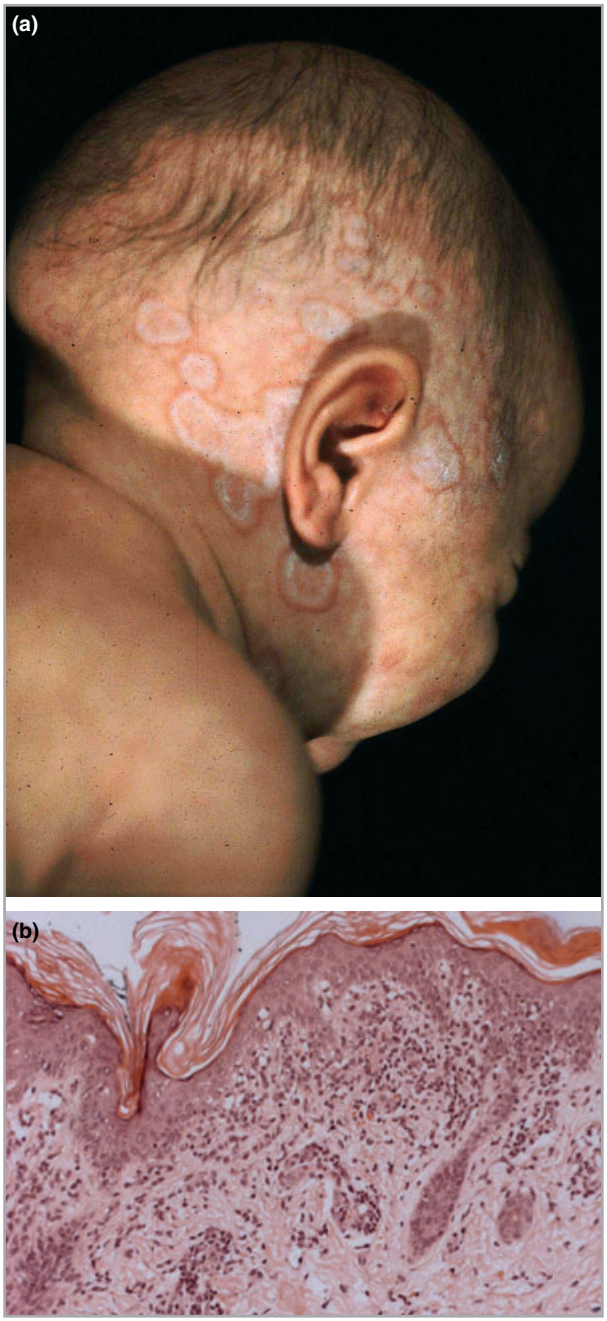

Fig 1. (a) Clinical appearance of head lesions: inflammatory annular plaques, with hyperkeratotic borders and atrophic centres. (b) Histology of lesional skin. Note vacuolar degeneration at the dermal–epidermal junction and perivascular mononuclear infiltrate in the dermis (haematoxylin and eosin; original magnification × 250).

Serological and blood tests in the mother revealed anaemia, increased platelet count, elevation of erythrocyte sedimentation rate and positivity for antinuclear antibody (ANA) with a speckled pattern (1 : 640). Anti-Ro/SSA and anti-La/SSB antibodies were positive on enzyme-linked immunosorbent assay (Fenning, U.S.A.).

Male A was not investigated serologically. Females B (at 15 months), C and D (at 3 months) showed ANA positivity (1 : 160 to 1 : 320) in immunofluorescent assays with Hep2 cells. Infants B, C and D were all positive for anti-La/SSB antibodies and negative for anti-Ro/SSA. There was elevation of liver enzymes. Electrocardiograms did not reveal conduction defects. Cardiac ultrasonography showed structurally normal hearts.

Laboratory abnormalities gradually resolved over the following months. After 1 year all the children were negative for ANA and anti-Ro/La antibodies.

The literature is conflicting about the overall risk of an anti-Ro/La antibody-positive mother having a child affected by NLE.[2] The actual incidence of the individual clinical manifestations of NLE is debated, because most data derive from retrospective studies. Buyon *et al.*[3] reported a much higher prevalence of cardiac as compared with skin manifestations, but it is possible that cutaneous involvement, less severe or misdiagnosed, could be under-reported in their survey. Only about 10% of patients present with both cardiac and skin disease. Thrombocytopenia is present in 10–20% of patients and elevation of aminotransferases in 20–40%.[4] Cimaz *et al.* found that cutaneous NLE appeared to be 10 times more frequent than CHB (16% vs. 1·6% of infants).[2] Solomon *et al.* estimated the risk of recurrent NLE in successive pregnancies as 25%, with a recurrence rate of 19% for CHB.[5] NLE has also been described in twins and in triplets.[6,7] In these cases newborns are more often discordant as to the expression of NLE and, interestingly, discordance is found not only in heterozygotic, but also in monozygotic twins.[8,9]

This is the first description of cutaneous NLE to occur in four siblings. All the infants presented a clinical uniformity with exclusively cutaneous features, similar for type, distribution, course and in the absence of cardiac involvement. At the same time, females B, C and D showed au unusual serological profile, all being positive for anti-La/SSB antibodies (investigated concurrently with the mother). The negativity of anti-Ro/SSA antibodies in the children, and the positivity in the mother, could be explained by two hypotheses. The first is that, as reported,[10] there was a selective transplacental transfer of anti-La in the absence of the more common anti-Ro antibodies from mother to foetus. The second hypothesis is that the anti-Ro/SSA antibodies crossed the placenta and

accumulated only into the skin and liver, being undetectable in blood. This hypothesis could further demonstrate a different affinity of maternal antibodies to different fetal tissue antigens, and is able to explain the different clinical presentations of NLE. Both the suggested hypotheses should be investigated in order to understand the concordance between clinical and serological aspects.

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Anastrozole-induced subacute cutaneous lupus erythematosus

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Sir, We describe a 73-year-old woman who was referred to our department for an intense cutaneous eruption. She had recently had breast cancer, which had been treated with surgery 7 months previously, then with radiotherapy, followed by anastrozole. She also had a high arterial blood pressure, treated for many years with an angiotensin-converting enzyme (ACE) inhibitor.

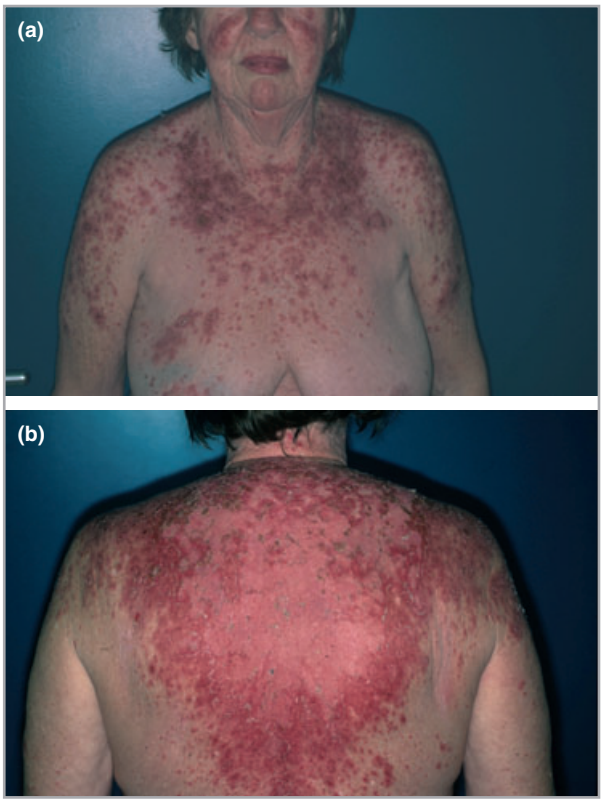

Fig 1. Annular and papulosquamous lesions on (a) the anterior chest and (b) the back.

The cutaneous eruption had begun 1 month after the beginning of anastrozole treatment. This treatment was discontinued and the ACE inhibitor was stopped and replaced with atenolol. The eruption improved slightly. Five weeks later, anastrozole was reintroduced and the eruption reappeared with greater intensity.

This eruption affected the upper trunk, neck, face, and extensor surfaces of the upper arms (Fig. 1). Lesions were annular, polycyclic and erythematosquamous, with a desquamative collarette. The patient had an intense burning sensation and pruritus. Clinical examination was otherwise normal; in particular, there was no oral ulceration.

A biopsy, performed on the upper back, showed an atrophic epidermis with basal vacuolar change. Dermal oedema and a lymphocytic inflammatory infiltrate were present. There was no renal involvement. Autoimmune screening showed speckled pattern antinuclear antibodies, positive anti-Ro/SSA antibodies and positive anticardiolipin antibodies. Antihistone antibodies and double-stranded DNA antibodies were negative. We therefore made a diagnosis of subacute cutaneous lupus erythematosus (SCLE).

We concluded that the SCLE had been induced by anastrozole. Anastrozole was stopped and the patient was treated with hydroxychloroquine and topical corticosteroids. Complete