gland tumors, lymphangiomas, hemangiomas, neuromas, neurofibromas, schwannomas, adenocarcinomas, ectopic thyroid tissue, or thyroglossal duct remnants.[3,4]

Many of the aforementioned lesions are similar in clinical appearance and can only be differentiated by means of histologic evaluation. Traditional imaging techniques (simple x-rays, computed tomography scans, magnetic resonance) are often altered by dental implants or fillings. Tongue lesions are therefore frequently removed on the basis of the findings of physical examination alone.[5]

The generalized use of dermatologic ultrasound in the last 10 years has included the inspection of mucosal tissues, but few cases extending this application have been reported in dermatology journals. All such descriptions available have been published in journals of oral medicine or maxillofacial surgery. The lesions described are usually oval, well-defined but not encapsulated, and heteroechoic (iso- or hypoechoic with respect to surrounding structures). They have absent or very weak Doppler signals (Table 1). There might be slight posterior acoustic enhancement. Short, parallel hyperechoic bands are less often seen in these lipomas than in those at other locations.[2,5]

A lesion on the tongue is examined in much the same way as lesions elsewhere. The anterior two-thirds of the tongue can usually be examined without assistance, but given the limited space in the mouth it may be useful to use a smaller or ''hockey-stick'' probe to facilitate mobility. A protective shield is necessary when examining mucosal tissue, and the patient should be informed that the gel used for dermatologic ultrasound is nontoxic.

Ultrasound imaging can help the dermatologist evaluate a lesion's size, shape, degree of vascularization, and attachment to other structures and should be considered for the differential diagnosis of tongue lesions.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Daylight Photodynamic Therapy in the Treatment of Actinic Keratosis in Carriers of Oculocutaneous Albinism: Report of Three Cases☆

Terapia fotodinámica con luz de día en el tratamiento de queratosis actínicas en pacientes portadores de albinismo oculocutáneo: presentación de 3 casos

To the editor:

Oculocutaneous albinism (OCA) leads to hypopigmentation of the skin, hair and eyes .[1] The most severe phenotype, OCA1, is characterized by the complete lack of melanin production; in subtypes, OCA2, OCA3, and OCA4, some pigment production occurs over the years.[2]

Actinic keratosis (AK) are premalignant lesions of the skin, commonly located in areas exposed to ultraviolet (UV) radiation.[3] OCA patients have an exaggerated sensitivity to UV radiation, which leads the onset of AK lesions and squamous-cell carcinomas, even at young age.

In daylight PDT (DL-PDT), the activation of the photosensitizer protoporphyrin IX (PpIX) from the methylaminolevulinate (MAL) cream by visible light, allows the treatment of AK with less adverse effects of pain and erythema ,[4] which favors its use as an alternative to conventional PDT in patients with excessive photosensitivity.[5] Other therapeutic options focused on the field of cancerization, such as imiquimod and 5-fluorouracil creams require long-term home treatment regimens and may lead to intense local skin reactions while, ingenol mebutate gel is usually limited to areas of up to 25 cm^2 . Thus, the treatment of AK with DL-PDT presents a number of advantages in patients with OCA, in comparison with other treatments.

In 2015, we treated the first patients with OCA at Dona Libânia Dermatology Center in Fortaleza, Brazil. There were

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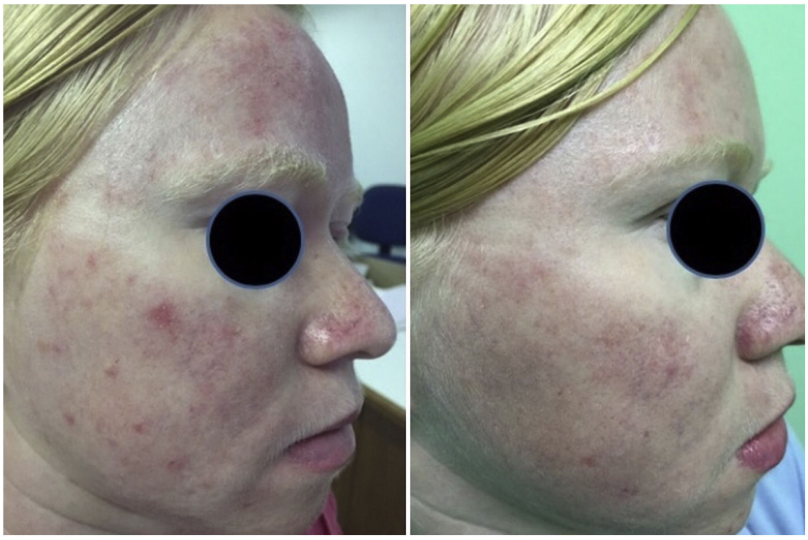

Figure 1 Clinical improvement after 6 months of DL-PDT.

three female patients, aged 22, 48 and 65 years old. The use of DL-PDT in patients with OCAs was approved by the local ethics committee.

On the day of the procedure, after the curettage of facial AK lesions, a chemical filter with sun protection factor (SPF) 50 plus lotion (Actinica®, Galderma, France) was applied on the face and other exposed areas. Subsequently, approximately 1 g of of the 16% MAL cream (Metvix®, Galderma, France) was applied throughout the face, and patients stayed outdoors, in the shade, between 7:30 and 9:30 AM. After that, the MAL cream was removed, and patients were discharged, with the instruction to strictly maintain the use of sunscreen at home. AK lesions were counted before the session and after 4, 12 and 24 weeks of follow-up. The patients were also assessed, after 2 hours of exposure, for the occurrence and grade of local skin reactions.

A total of 66 facial AK lesions were treated in the three patients, all of whom completed the 24-week period of follow-up. Of the 66 treated lesions, 52 lesions (78.8%) were considered as clinically cured at weeks 4 and 12, and 50 lesions (75.8%) were considered as cured at week 24 (Figures 1 to 3). No pain or burning sensation was reported by any of the patients during the 2-hour shade period. The 22-year-old patient was the only one who presented moderate facial erythema immediate after the procedure, with

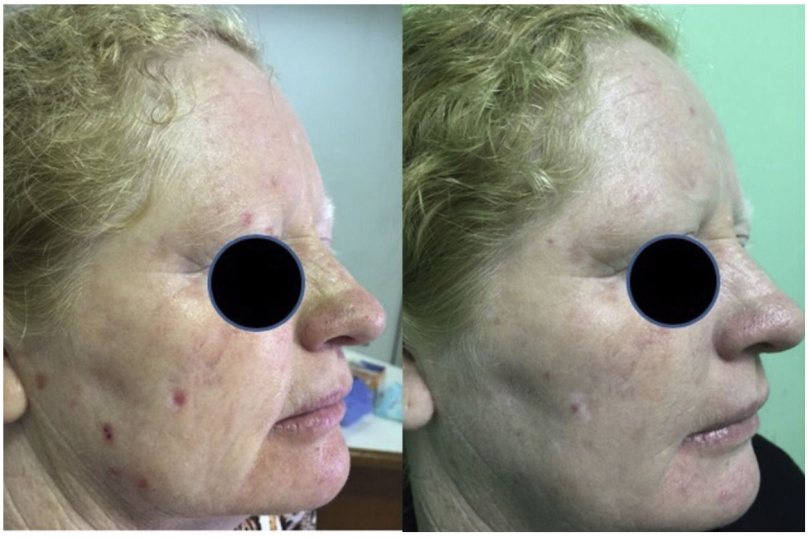

Figure 2 Field cancerization of the skin before and 6 months after DL-PDT session.

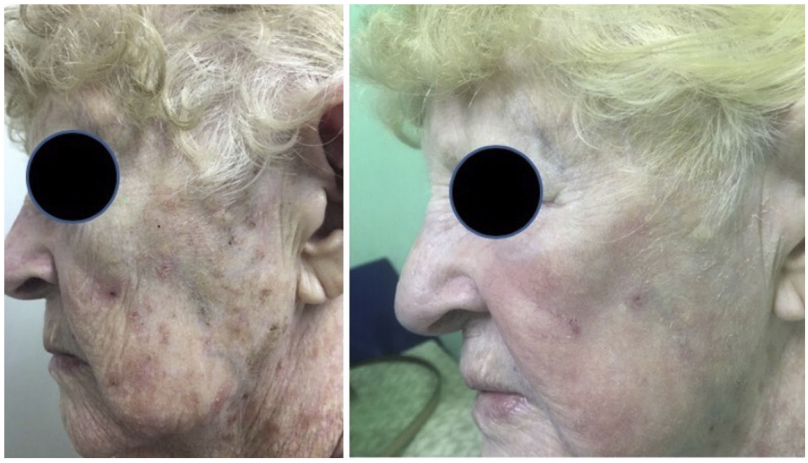

Figure 3 Treatment area before and after 24 weeks of follow up.

mild pruritus during outdoor exposure (Figure 4). However, she reported being exposed to the sun for about 10 minutes during the 2-hour period with MAL cream. None of the three patients had adverse effects at the 4-week follow-up visit.

In the current study, we found that nearly 76% of the treated AK lesions could be considered cured after 6 months of follow-up. This cure rate is within the range found in the major European[6] and Australian[7] muticenter studies of DL-PDT. Since patients with OCA present with extensive AK, with a large number of lesions at an early age, it is important to develop alternative tratments that rapidly eliminate the lesions in large areas and with little local reaction.

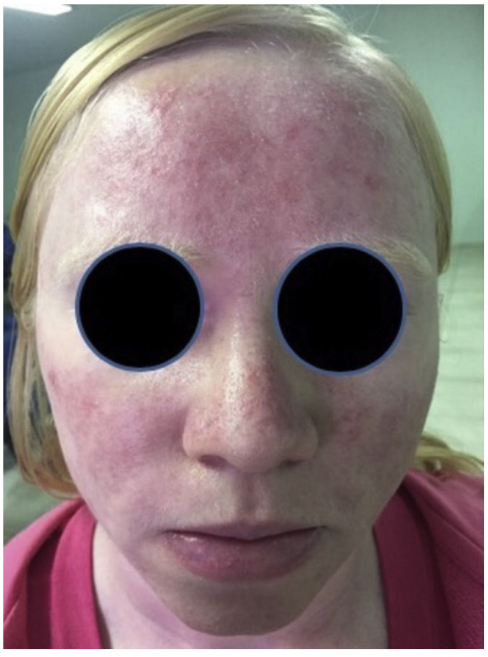

Figure 4 Moderate facial erythema immediately after the 2-hour outdoor exposition.

During DL-PDT, avoidance of exposure to direct UV radiation through permanence in the shade during the whole period of 2 hours is fundamental in patients with OCA. We observed moderate erythema immediately after the procedure in one of the patients, who, despite orientation, was exposed to sunlight for about 10 minutes. The city of Fortaleza is located at a low latitude (03°43'02'' South), with average annual temperature of 26.3 °C. Such warm and sunny weather characteristics allow the use of DL-PDT throughout the year. On the other hand, patients with OCA undergoing DL-PDT in places with similar characteristics have to be carefully instructed about the procedure. Moreover, the use of SPF50 clothing and sunscreen is recommended during the 2-hour exposure in the shade. In our three patients, DL-PDT sessions were scheduled and performed early in the morning, with no overcast or rainy weather.

In conclusion, DL-PDT may be an option in the treatment of multiple lesions of AK on the face of patients with OCA. One of the advantages of treating a wide area such as the face in a single day is the ability to maintain the daily activities of these patients, especially when compared with treatments that require a length of time at home and which may cause skin reactions, both of which can impair adherence to treatment.

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Plaque Psoriasis Flare and Peripheral Edema in a Patient Treated With Atezolizumab☆

Brote de psoriasis en placas y edema periférico en un paciente tratado con atezolizumab

To the Editor:

A 75-year-old man was referred to the dermatology clinic for evaluation of skin lesions after receiving his first dose of atezolizumab (1200 mg) for treatment of poorly differentiated stage IV urothelial carcinoma of the prostatic urethra (metastasis to the lungs, liver, bone, and lymph nodes). Twelve months previously, the patient had completed systemic treatment with chemotherapy (cisplatin 75 mg/m^2 day 1 + gemcitabine 1250 mg/m^2 on days 1 and 8 and every 21 days) in 4 cycles. No clinical or radiological response was observed, and the treatment was poorly tolerated (asthenia, vomiting, and moderate kidney failure). He had a 42-year history of mild psoriasis affecting the elbows and knees.

Twelve days after the first infusion of atezolizumab, his existing lesions began to worsen, and new lesions began to appear on the extensor aspects of both limbs and the trunk. These were intensely pruritic and compatible with a clinical diagnosis of psoriasis (Psoriasis Area Severity Index = 12, body surface area affected = 6, visual analog scale for itching = 10). These findings were accompanied by distal edema affecting both lower limbs. The patient was prescribed oral prednisone 30 mg for 7 days to be tapered by 10 mg every week, bilastine 20 mg every 12 hours, and topical treatment with clobetasol propionate 0.1% cream every 12 hours for 15 days. His lesions resolved in 3 weeks, and no residual lesions were observed. Atezolizumab was discontinued because of the skin toxicity. Given the patient's poor general status, he refused all cancer medication and is currently receiving palliative care. He was diagnosed with probable adverse reaction to atezolizumab (Naranjo algorithm, 6 points).

Programmed death 1 (PD-1) is a key immune checkpoint receptor that is expressed on T cells and functions mainly in peripheral tissue.[1] Atezolizumab is the first PD ligand 1 (PD-L1) inhibitor approved by the United States Food and Drug Administration. This human immunoglobulin G1 monoclonal antibody binds selectively to PD-L1 and prevents interaction with PD-1 and B7-1 (also known as CD80), while sparing the interaction between PD ligand 2 (PD-L2) and PD-1.[2] When PD-1 is activated, the immune system is inhibited, thus enabling tumor growth. In their various indications for different cancers, new anti–PD-1 drugs (nivolumab, pembrolizumab, pidilizumab) and anti–PD-L1 drugs (atezolizumab and durvalumab) curb this inhibition by enabling the immune system to control tumor progression.[1]

There have been various reports of adverse reactions to PD-1 and PD-L1 inhibitors in up to 50% of patients. These were mainly cutaneous (lichenoid reactions, eczema, vitiligo, and pruritus) and mild and did not require treatment to be discontinued.[3,4] However, other authors report these inflammatory reactions to be severe, requiring treatment with oral corticosteroids, and highlight an objective antitumor response.[5] One group of cutaneous reactions to this drug are those based on neutrophils, which, owing to their increased count in the skin, cause Sweet syndrome, acute generalized exanthematous pustulosis, intracorneal pustular drug eruption, and psoriasis.[6] Other, less common cutaneous adverse reactions include actinic keratosis, squamous cell carcinoma, and seborrheic keratosis.[3] Peripheral edema is an adverse reaction that affects 10% of patients receiving treatment with atezolizumab.[7]

Cases of psoriasis triggered or exacerbated by this drug family are starting to be reported, although the condition

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