

Erythrodermic Presentation of Atopic Dermatitis in a Patient with Secondary Adrenal Insufficiency Caused by Oral Glucocorticosteroid Abuse

Dear Editor,

A 41-year-old man presented to the Department of Dermatology for the first time due to an exacerbation of atopic dermatitis (AD) in the form of erythroderma.

The patient had a history of atopic diseases, with being AD active from infancy. On clinical examination, generalized erythematous skin lesions causing acute pruritus and accompanied by severe skin exfoliation and dryness were present. On closer examination, the patient had a collection of signs and symptoms characterizing Cushing syndrome that included a round and full face ("moon face"), supraclavicular fat pads, and proximal muscle atrophy.

The patient stated that AD had exacerbated six years earlier. He had received systemic treatment consisting of methotrexate followed by cyclosporine in another medical facility. However, both medications had proven ineffective and caused malaise. Only oral glucocorticosteroids had proven successful. The patient had been satisfied with the quick and observable effects, and, as he stated, he refrained from regular dermatological visits for six years. During that time, he consistently took 4 mg of methylprednisolone twice daily.

Laboratory tests showed undetectably low levels of cortisol, triacylglycerols (TAG) at 288 mg/dL, and



Figure 1. The patient with atopic dermatitis (AD) before the introduction of dupilumab in the left picture. He presented with generalized erythematous skin lesions accompanied by skin exfoliation and dryness. In the image on the right, the patient is shown after 7 months of dupilumab treatment.

total cholesterol levels (CHC) of 81 mg/dL. Based on laboratory findings, clinical presentation, and histopathological evaluation of the skin biopsy, the diagnoses were secondary adrenal insufficiency caused by oral glucocorticosteroid abuse and AD in the form of erythroderma.

The endocrinologist suggested a progressive reduction of the dose of methylprednisolone, starting at 2 mg twice daily. Total and sudden drug withdrawal was unacceptable, as it could cause an adrenal crisis. Methylprednisolone was eventually discontinued after being administered for 5 months while the blood levels of ACTH, cortisol, ionized sodium, and ionized potassium were monitored every 4 weeks. 25 mg of hydrocortisol in divided doses was the actual treatment for adrenal insufficiency, with plans to also gradually reduce the dose. Since the commencement of endocrinological treatment, the dose was reduced to 15 mg after 5 months and to 10 mg after 7 months. Following an 8-month period, the patient began taking 10 mg as needed, usually a few times each month. Calcium carbonate in a dose of 1000 mg taken once daily before a meal for 5 months and vitamin D3 protected the patient from osteoporosis, another manifestation of Cushing syndrome. An initial dose of 4000 IU was prescribed. It is vital to emphasize that all dose adjustments in the endocrinological treatment of Cushing syndrome were a direct consequence of laboratory testing that was performed.

In terms of erythrodermic AD management, the patient was treated with cyclosporin, which was once again ineffective. The patient was then prepared for the introduction of dupilumab. A 300 mg dose of the medication was subcutaneously administered every 2 weeks for over a year with positive outcomes, with an initial dose of 600 mg. The patient developed gynecomastia at the beginning of the treatment, initially categorized as another manifestation of Cushing syndrome. However, due to its unilateral nature, it was later identified as a benign adverse event of dupilumab, as described in the literature (1). Due to a decline in effectiveness, the treatment was recently switched from dupilumab to baricitinib, with positive outcomes.

Erythroderma, which the patient presented in our case, is an acute condition characterized by erythema and scaling that involves more than 90% of the skin's surface area (2,3). It can be potentially fatal due to electrolyte imbalance, fluid loss from capillary dilation, and significant heat dissipation (3). According to estimates, erythroderma is relatively rare, affecting approximately 1-2 patients for every 100,000 people per year, with AD comprising 8.7% of all cases

of erythroderma (2,4). Despite growing therapeutic possibilities for AD, corticosteroids remain the drug of choice in severe exacerbations, including erythroderma, when we cannot afford to wait for the effects of therapy.

Oral glucocorticosteroids can be an effective treatment for acute flares of AD (5). However, there is a lack of evidence for the long-term efficacy and safety of oral glucocorticosteroids in the treatment of AD (5). Reported side-effects include endocrine disturbances, gastric ulcers, cardiovascular disorders (arterial hypertension, atherosclerotic disease), osteoporosis, glaucoma, cataracts, and an increased risk of infections. Corticosteroids also have an undesired action on the skin that can result in steroid acne, skin atrophy, striae, telangiectasias, hypertrichosis, and impaired wound healing. The psychological adverse effects of steroid treatment can be quite severe and include depression and psychosis (6). The therapy should only be applied in the short-term, not exceeding one week, due to the occurrence of the above-mentioned side-effects, which presented in as Cushing syndrome our patient (5).

However, glucocorticoids are one of the most commonly used drugs in clinical dermatology practice, raising concerns about the risk of their misuse, which can lead to secondary adrenal insufficiency, among other complications (7). When no other treatment options are available, it should be noted that many of the side-effects of oral glucocorticosteroids can be mitigated through close monitoring and the implementation of appropriate preventive measures (7).

Patient consent form

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that names and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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