A chronic nonresponsive facial eruption

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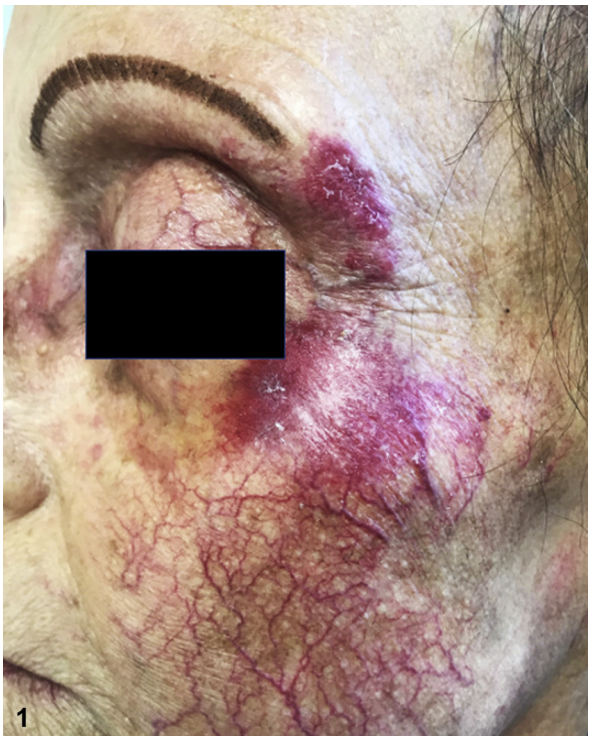

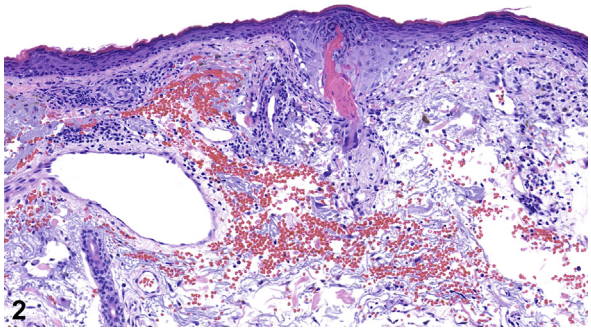

A 67-year-old woman presented for evaluation of a waxing and waning facial eruption, which had been present for several years. Of note, the patient denied any prior use of topical steroids. The patient took omeprazole daily for years but denied any correlation with her cutaneous eruption. Physical examination revealed erythematous atrophic plaques and thin plaques with scale on the bilateral cheeks within a diffuse background of telangiectasia (Fig 1). Dermoscopic evaluation did not reveal follicular plugging. Prior laboratory evaluation by her primary care physician demonstrated absence of antinuclear antibodies and anti-dsDNA antibodies, and low levels of C3 and C4. A punch biopsy revealed cell-poor interface dermatitis with scattered apoptotic keratinocytes, along with superficial perivascular and perifollicular lymphoplasmacytic infiltrate (Fig 2).

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Question 1: Which of the following is the best diagnosis?

A. Actinic purpura

B. Allergic contact dermatitis (ACD)

C. Erythematotelangiectatic rosacea

D. Subacute cutaneous lupus erythematosus (SCLE)

E. Topical corticosteroid-induced cutaneous atrophy

Answers:

A. Actinic purpura — Incorrect. Actinic purpura presents with violaceous macules and patches on photoaged skin. Lesions are commonly found on the face, neck, and arms, specifically on the extensor surfaces of the forearms and the dorsal aspects of the hands. The histopathologic features of actinic purpura include extravasation of red blood cells within the dermis and solar elastosis.[1]

B. Allergic contact dermatitis (ACD) — Incorrect. ACD typically presents with erythema and pruritus. Acute ACD can present with vesiculation or bullae, while chronic ACD can present with lichenification. The affected areas are localized to the area of allergen contact. The histopathologic features of acute ACD include edema and intraepidermal vesiculation without epidermal hyperplasia. Subacute-to-chronic ACD presents with epidermal hyperplasia. Eosinophils are present in all stages of ACD, and eosinophilic spongiosis is a variable feature.[2]

C. Erythematotelangiectatic rosacea — Incorrect. Erythematotelangiectatic rosacea presents with episodic flushing, dilated telangiectasia, and central facial erythema. Erythema may also affect the peripheral face, ears, neck, and upper aspect of the chest. The histopathologic features of erythematotelangiectatic rosacea are telangiectasia with lymphohistiocytic perifolliculitis, vascular ectasia, and solar elastosis.[3]

D. Subacute cutaneous lupus erythematosus (SCLE) — Correct. SCLE typically presents with erythematous plaques, which may have an annular configuration or papulosquamous appearance. Lesions occur on sun-exposed areas with sparing of the midfacial skin. The sides of the face, upper trunk, and extensor aspects of the upper extremities are commonly affected. Hypopigmentation typically results after lesion resolution, but SCLE does not lead to scarring or dermal atrophy. Histopathologic hallmarks of SCLE include a cell-poor (vacuolar) interface dermatitis, epidermal atrophy or hyperplasia, superficial perivascular and periadnexal lymphocytic inflammation, and mucin deposition.[4]

E. Topical corticosteroid-induced cutaneous atrophy — Incorrect. Cutaneous atrophy secondary to chronic topical corticosteroid use presents with thinning, telangiectasia, and small hematomas. Advanced stages present with lacerations and hemorrhage in the dermis, which can ultimately lead to dissecting hematomas. The histopathologic features of topical corticosteroid-induced cutaneous atrophy are thinning of the epidermis and telangiectasia.[5]

Question 2: Which of the following represents the most appropriate initial therapeutic plan?

A. Discontinuation of topical corticosteroid application

B. Drug cessation, photoprotection, topical corticosteroids or topical calcineurin inhibitors, and antimalarials

C. Photoprotection and topical vitamin C

D. Systemic corticosteroids

E. Topical metronidazole and photoprotection

Answers:

A. Discontinuation of topical corticosteroid application — Incorrect. In the setting of topical corticosteroid-induced atrophy, discontinuation of topical corticosteroid use has been shown to reverse cutaneous atrophy.[5]

B. Drug cessation, photoprotection, topical corticosteroids or topical calcineurin inhibitors, and antimalarials — Correct. These are treatment options for non-drug-induced SCLE and drug-induced SCLE. For localized disease, topical corticosteroids or calcineurin inhibitors can be utilized. Photoprotection is crucial, with reapplication of sunscreen advised every 2 hours. Antimalarials are the initial systemic therapy of choice for cutaneous lupus erythematosus, with hydroxychloroquine as the preferred agent. In antimalarial-resistant cases, methotrexate, thalidomide, or mycophenolate mofetil can be utilized.[4] However, for patients with SCLE potentially induced by medication, trial withdrawal represents an appropriate initial step in management. Approximately 30% of cases of SCLE are drug induced. Non-drug-induced SCLE and drug-induced SCLE present with identical clinicopathologic features and can only be distinguished by response to drug withdrawal. The majority of drug-induced SCLE cases usually demonstrate antinuclear antibodies (64%) and anti-Ro(SS-A)

positivity (74%), with a lower proportion testing positive for anti-La(SS-B) (25%), anti-dsDNA (21%), and anti-histone (17%) antibodies.[4] In our case, it is possible that that SCLE was drug-induced (omeprazole), but we cannot say this for certain, as antimalarial therapy was initiated at the same time as drug cessation.

C. Photoprotection and topical vitamin C — Incorrect. These are treatment options for actinic purpura. Photoprotection is recommended as a preventative measure. Topical vitamin C may reduce purpura without significant adverse effects.[1]

D. Systemic corticosteroids — Incorrect. While systemic corticosteroids may be utilized to treat systemic lupus erythematosus, they are not the most appropriate initial therapeutic plan for cutaneous lupus erythematosus.

E. Topical metronidazole and photoprotection — Incorrect. The described treatment is appropriate for erythematotelangiectatic rosacea. Other treatment options include topical azelaic acid, topical vasoconstrictors, and laser therapy.[3]

Question 3: Which of the following represents a common trigger of SCLE?

A. Prednisone

B. Nickel

C. Minocycline

D. Proton pump inhibitors

E. Hydroxyurea

Answers:

A. Prednisone — Incorrect. Chronic prednisone therapy may result in increased purpura, particularly in the setting of photodamage.

B. Nickel — Incorrect. Nickel is one of the most common causes of ACD.[2]

C. Minocycline — Incorrect. Minocycline is a common cause of drug-induced systemic lupus erythematosus, not drug-induced SCLE.[4]

D. Proton pump inhibitors — Correct. In a recent study, proton pump inhibitors proved to be the most common culprit among 88 patients with drug-induced SCLE. Some of the other implicated drug classes include antihypertensives, antifungals, chemotherapeutics, statins, antiepileptics, and biologics.[4]

E. Hydroxyurea — Incorrect. Hydroxyurea is a cause of drug-induced dermatomyositis, not drug-induced SCLE.

Abbreviations used:

ACD: allergic contact dermatitis
SCLE: subacute cutaneous lupus erythematosus

Conflicts of interest

None disclosed.

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