A Less Known Dermatological Emergency

A 22-year-old Chinese lady with a history of discoid eczema presented with a weepy, vesicular facial eruption of 4 days' duration. She had been prescribed a course of oral amoxicillin clavulanate by her general physician without any significant improvement in her condition.

On physical examination, she was febrile with a temperature of 38.5°C. There were extensive vesicular lesions with erosions and yellow adherent crusts over the entire face (Fig. 1). The trunk and limbs were less extensively involved and comprised predominantly of multiple monomorphic eroded vesicles in a punched out configuration (Figs. 2 and 3). Cervical lymphadenopathy was present bilaterally. Ophalmologic examination under slit-lamp was normal.

What is the diagnosis?

(A) Impetigo

(B) Severe flare of atopic dermatitis

(C) Eczema herpeticum

(D) Varicella infection

(E) Stevens-Johnson syndrome/Toxic epidermal necrolysis

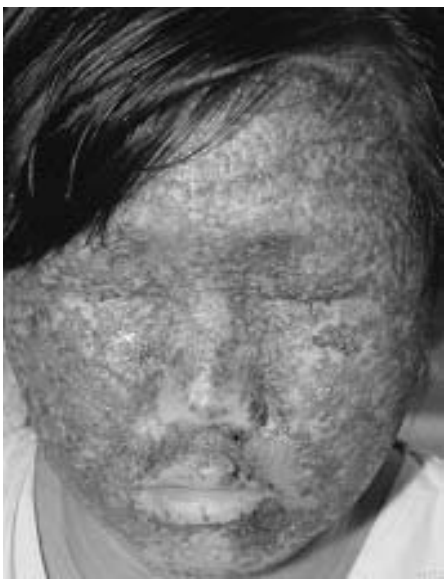

Fig. 1. Extensive tense vesicles on the face which had broken down to form crusted erosions.

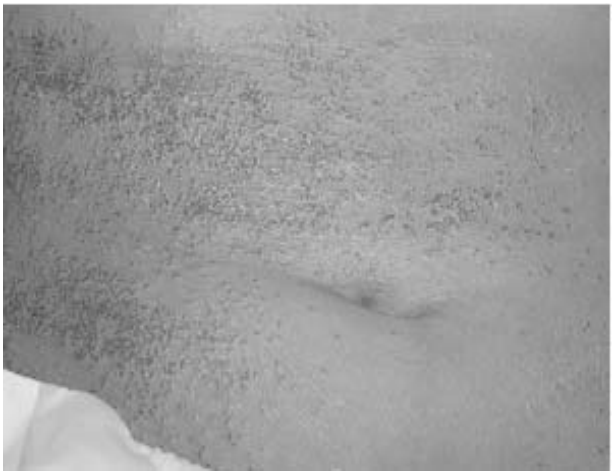

Fig. 2. An area of extensive early erosions over the abdomen.

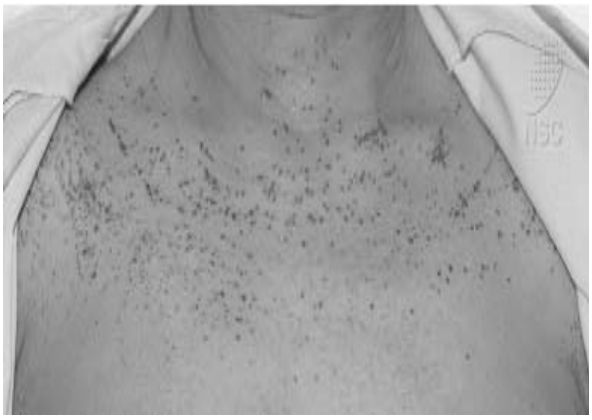

Fig. 3. Further-evolved erosions on the chest demonstrating their "punctuate" nature with the absence of significant oedema.

Discussion

A clinical diagnosis of eczema herpeticum was made and the patient was started on intravenous acyclovir. Wound swabs of the bases of eroded vesicles were sent for immunofluorescence antibody staining; polymerase chain reaction demonstrated the presence of herpes simplex virus type 1 (HSV-1). Significant clinical improvement was observed over the course of 1 week.

Eczema herpeticum, also known as Kaposi's varicelliform eruption, was initially described by Moriz Kaposi in 1887. It results from widespread HSV infection of damaged skin. It is most commonly associated with atopic dermatitis, but

Answer: (C)

other skin disorders such as contact dermatitis, seborrheic dermatitis, psoriasis, pemphigus foliaceous, pemphigus vulgaris, pityriasis rubra pilaris and cutaneous T-cell lymphoma have also been reported.[1] The reason for these skin diseases being predisposed to the infection is unknown but a deficiency of cathelicidins in the skin may be a contributing factor.[2]

Eczema herpeticum typically presents several days after initial exposure to the virus. Large numbers of vesicles form over active or recently healed skin lesions with a predilection for the face. Lesions appear in crops for several days more before becoming pustular and umbilicated. Systemic features of fever and lymphadenopathy are also commonly present.

The diagnosis of eczema herpeticum hinges primarily on a high index of suspicion and treatment should not be delayed pending the results of laboratory tests. An important clue to the diagnosis is a past history of atopic dermatitis, and rarely other skin diseases. Clinically, eczema herpeticum may present with considerable diagnostic confusion. Vesicles and erosions can be present in both a flare of atopic dermatitis as well as eczema herpeticum. In the former, vesiculation occurs secondary to the marked spongiosis at the histological level and oedema is thereby expected to be significantly present in the lesions clinically. In the latter, vesiculation occurs secondary to acantholysis (breakdown of the connections between keratinocytes at the microscopic level) and the lesions are thereby more localised and "punctate", and are not associated with significant oedema. In addition, patients with eczema herpeticum tend to have fever and are more systemically unwell than patients with an uncomplicated flare of atopic dermatitis.

Supportive laboratory investigations include Tzanck smear and HSV cultures and polymerase chain reaction. These tests, however, are limited by a uniform lack of sensitivity and thus treatment should not be halted based on negative results, especially if the clinical suspicion is high. Skin biopsy can also be helpful in identifying the microscopic features of herpes infection, which include ballooning degeneration of keratinocytes, multinucleated giant cells and peripheral margination of nucleoplasm with basophilic rimming of nuclei.

The cornerstone of management of eczema herpeticum is a systemic antiviral, of which intravenous acyclovir is the most commonly used agent. Valacyclovir has also been shown to be effective and has the benefit of a greater bioavailability and a more convenient dosing schedule. The benefits of these antiviral agents are greatest when they are administered early in the disease to limit the damage caused by the virus. Systemic antibiotic therapy is also important as lesions are often impetiginised and the resultant bacteremia is the major cause of mortality in patients with eczema herpeticum. Generally, this entails the use of a second generation oral cephalosporin such as cephalexin. The patient's fluid and electrolyte status should also be monitored closely, especially if the lesions are more extensive. Adjuvant therapies, including sedative antihistamines for pruritus, anti-pyretics for fever and wet compresses for weepy lesions, are also helpful.

Eczema herpeticum ranges in severity from mild to fatal, with mortality rates of up to 75% if systemic antiviral therapy is not administered.[3] In patients with mild to moderate disease, complete resolution is expected within 2 to 6 weeks. In more severe disease, however, the lesions may heal with scarring. Eczema herpeticum, like herpes infection, can be recurrent but subsequent episodes tend to be milder and are not associated with systemic features.

REFERENCES

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