

A diffuse painful desquamating rash

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CASE REPORT

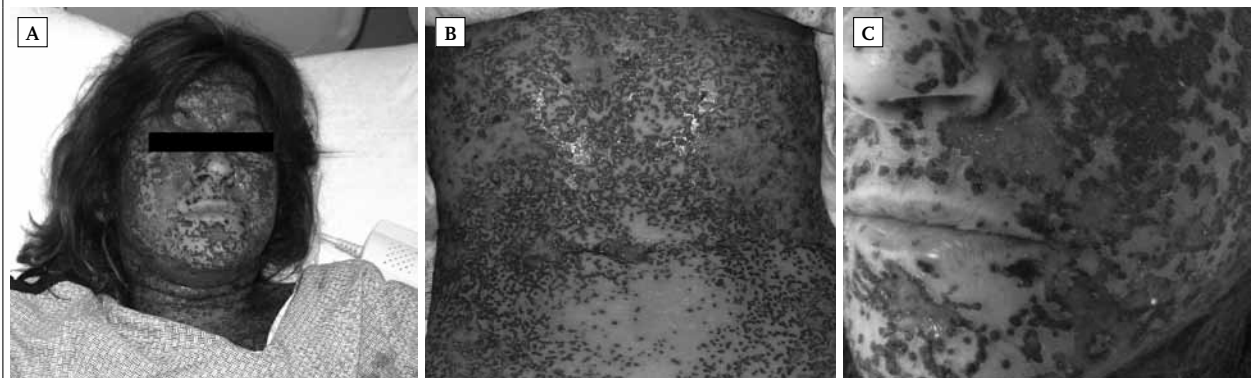
A 44-year-old woman was transferred to our hospital service for evaluation and treatment of a generalised, painful rash, with fever and lymphadenopathy present for ten days (*figure 1*). Prior to transfer, our patient spent a week at an outside hospital where she was treated with multiple antibiotics, including bactrim, vancomycin, nafcillin, and ceftazadine. She also had received intravenous solumedrol. Despite treatment, the patient's rash, initially localised to the face, rapidly progressed. On admission to our hospital, we performed a punch biopsy

of skin from the patient's trunk and submitted it for frozen and permanent sections. Our patient's past medical history included severe atopic dermatitis treated with prednisone 10 mg daily and multiple MRSA infections of the soft tissue and lung.

WHAT IS YOUR DIAGNOSIS?

See page 374 for the answer to this photo quiz.

Figure 1. *Clinical appearance of the patient*



The lesions initially involved the face and consisted of erythematous vesicles and ulcers. B) The process rapidly spread to her trunk and proximal extremities and became confluent, resulting in large areas of desquamation. C) Representative lesions on the face demonstrated a mixture of discrete crusted ulcers and confluent desquamation.

DIAGNOSIS

Our patient's history of antibiotic use and a rapidly progressive rash with large eroded areas prompted us to submit tissue for frozen section to rule out Stevens-Johnson syndrome. Examination of the frozen section slides revealed massive epidermal necrosis and multinucleated keratinocytes with nuclear chromatin margination and moulding (*figure 2A and B*) consistent with a diagnosis of eczema herpeticum (EH). Treatment with intravenous acyclovir was initiated and the patient dramatically improved over the course of the next week. She was also treated with intravenous clindamycin for any potential bacterial superinfection and topically with emollients and vaseline-impregnated gauze. Our initial diagnosis was subsequently confirmed by direct fluorescent antibody, which was positive for herpes simplex virus type 1 (*figure 2C*).

Patients with eczematous skin disease or atopic dermatitis are prone to the development of viral skin infections. Among the most commonly recognised is eczema herpeticum. EH is caused by dissemination of herpes simplex virus, typically herpes simplex type 1.¹ It often presents in the first three decades of life. According to a study of 100 EH patients by Wollenberg *et al.*, 20% of the cases were related to primary infection, 26% were caused by secondary infection and the remaining 54 cases could not be characterised.² Rates of recurrence have been reported to be as low as 16% to as high as 50%.^{1,2}

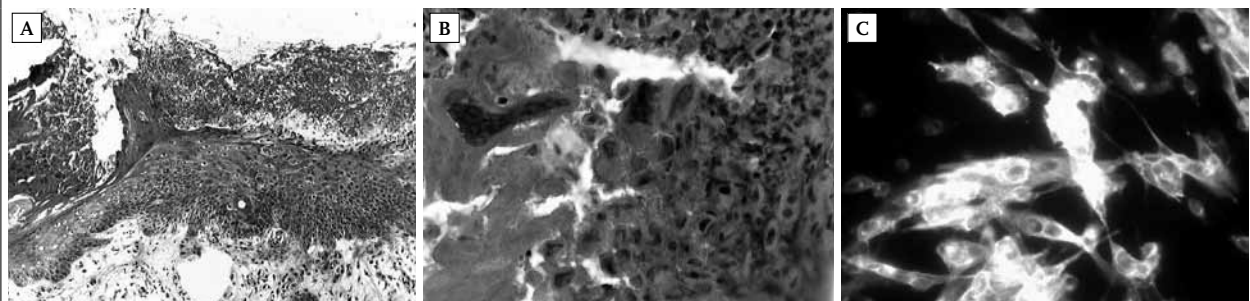
Clinically, the disease is characterised by monomorphic, dome-shaped vesicles that develop into punched-out,

crusted erosions. The eruption disseminates over 7-10 days most commonly affecting the head, neck and upper body. Patients may also experience fever, malaise and lymphadenopathy. Feared complications include keratoconjunctivitis, viraemia, meningitis, and encephalitis.^{1,3} The diagnosis of EH is based on the clinical presentation and a high clinical suspicion should prompt immediate treatment with IV acyclovir or oral valtrex for seven days.⁴ The diagnosis should be confirmed by viral detection. Methods of detection include Tzank test, viral culture, light or electron microscopy, direct fluorescent antibody and PCR.³ This case highlights the importance of early recognition of eczema herpeticum and the utility of submitting tissue for frozen section to facilitate a rapid diagnosis.

REFERENCES

1. Beck LA, Boguniewicz M, Hata T, Schneider LC, Hanifin J, Gallo R, et al. Phenotype of atopic dermatitis subjects with a history of eczema herpeticum. *J Allergy Clin Immunol.* 2009;124(2):260-9, 269.e1-7.
2. Wollenberg A, Zoch C, Wetzel S, Plewig G, Przybilla B. Predisposing factors and clinical features of eczema herpeticum: a retrospective analysis of 100 cases. *J Am Acad Dermatol.* 2003;49(2):198-205.
3. Bussmann C, Peng WM, Bieber T, Novak N. Molecular pathogenesis and clinical implications of eczema herpeticum. *Expert Rev Mol Med.* 2008;14;10:e21.
4. Wollenberg A, Wetzel S, Burgdorf WH, Haas J. Viral infections in atopic dermatitis: pathogenic aspects and clinical management. *J Allergy Clin Immunol.* 2003;112(4):667-74.

Figure 2. Histopathological features of a frozen section of a punch biopsy from the flank



Scanning magnification revealed massive necrosis of the epidermis (H&E, 100x). B) Higher power perspective demonstrated nuclear changes diagnostic of herpes infection, including margination of chromatin, multinucleation of keratinocytes, and moulding of keratinocyte nuclei (H&E, 400x). C) Direct fluorescence assay with a monoclonal antibody labelled with fluorescein isothiocyanate confirmed the diagnosis of eczema herpeticum and identified the virus subtype as HSV-1 (400x). H&E, haematoxylin and eosin.