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CE - MEDICAL ILLUSTRATION

Kaposi's varicelliform eruption

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A 25-year-old woman with hypothyroidism presented with 2 weeks of fever, chills, and fatigue, followed by a facial rash. She denied prior history of skin disorders. Physical examination revealed a temperature of 38.2 °C (100.8 °F), anicteric sclerae and a bilateral crusted malar rash involving the nasolabial folds (Fig. 1). Significant laboratory findings included marked elevations of the alanine transaminase 3,685 U/L (normal, 10–44 U/L), aspartate transaminase 4,101 U/L (normal, 14–40 U/L) and high International Normalisation Ratio (INR) 4.3, with slightly increased total bilirubin 2.4 mg/dL (normal, 0.1–1.0 mg/dL). The appearance of the facial rash was compatible with Kaposi's varicelliform eruption. With significant liver transaminase elevations, a provisional diagnosis of acute fulminant necrotising herpes simplex virus (HSV) hepatitis was made. Empiric therapy with intravenous aciclovir was immediately initiated. The blood HSV-2 polymerase chain reaction (PCR) assay returned 1.1 million copies/mL (normal, <50 copies/mL), confirming acute HSV-2 hepatitis. At the completion of 2 weeks of intravenous aciclovir therapy, the facial rash was substantially resolved and liver enzymes had almost normalised. She was discharged home with 4 weeks of oral valacyclovir.

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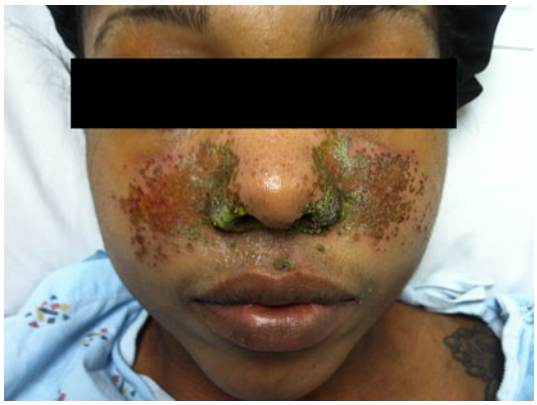

Fig. 1 Bilateral crusted and scaly skin rashes over her both malar cheeks and nasolabial folds

Kaposi's varicelliform eruption (KVE), also known as eczema herpeticum, is described as a monomorphic eruption of dome-shaped vesicles, most often on the face, head, neck, and trunk. The vesicles usually dry and form crusted papules within 2 weeks. It is a distinctive cutaneous rash associated most commonly with HSV (type 1 or 2) infection, and less often with vaccinia virus or coxsackievirus A16. KVE is usually superimposed on an inflammatory skin disorder, typically atopic dermatitis, seborrheic dermatitis or psoriasis [1].

HSV hepatitis ("anicteric hepatitis") is an uncommon clinical syndrome, representing 0.8 % of all causes of acute liver failure and 4 % of all viral causes of acute liver failure (Table 1) [2–5]. Given the rarity of this disease, our case highlights it is crucial for providing physicians to recognise KVE or other cutaneous herpetic lesions that could be important clinical clues to life-threatening HSV hepatitis, which has a >90 % mortality in the absence of appropriate antiviral therapy [1, 5]. Careful dermatological

Table 1 Selected studies of causes of acute fulminant hepatitis, focusing on acute viral hepatitis [2–5]

Studies	Center(s) involved	Total patients with acute hepatitis	Total patients with acute viral hepatitis	HSV hepatitis
Rakela et al. [2]	Single	34	23	2
Ostapowicz et al. [3]	17	308	36	0
Schiødt et al. [4]	19	354	43	1
Ichai et al. [5]	Single	360	100	5
Total	38	1056 (0.8 %)	202 (4 %)	8

examination can have a major impact on clinical diagnosis and treatment selection in patients with HSV hepatitis.

In conclusion, recognition of KVE is a critical clinical clue that should prompt providing clinicians to initiate potentially life-saving antiviral therapy while diagnostic confirmation is pending.

Conflict of interest Dr. Min declares no conflict of interest. Dr. Gnann is a consultant for Merck and GlaxoSmithKline.

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