

Painful skin eruption in a professional auto mechanic — a quiz

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A 37-year-old man, auto mechanic by trade, presented with multiple facial skin lesions, accompanied by burning sensation and pain, eye redness, fever and fatigue. The symptoms occurred 4 days before admission. Two weeks earlier he was consulted dermatologically due to hand eczema treated subsequently with topical steroids; additionally, he reported presence of concomitant facial erythema with scaling. Besides, the personal and familial medical history was unremarkable. Clinical examination revealed multiple skin erosions covered with serosanguineous crust, disseminated on his face, hands and forearms (Fig. 1); increased body temperature of 38.7 degrees Celsius, and conjunctival congestion. Laboratory tests revealed increased level of inflammatory parameters (C-reactive protein 21.25 mg/L, leukocytosis $13.48 \times 10^9/L$).

Which diagnosis is most likely in our patient?

- A. Impetigo
- B. Primary varicella infection
- C. Kaposi's varicelliform eruption
- D. Ecthyma
- E. Pemphigus vulgaris



Figure 1. Clinical presentation: multiple, disseminated facial skin erosions covered with serosanguineous crust

See next page for answer.

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ANSWER: KAPOSI'S VARICELLIFORM ERUPTION, ECZEMA HERPETICUM

Eczema herpeticum (EH), formerly known as Kaposi's varicelliform eruption, arises mostly as a secondary viral infection in patients with underlying skin disintegrity disorders. It was first described by Moritz Kaposi in the 19th century. EH is a rare condition, that responds well to antiviral therapy, but if disregarded, it can assume a fulminant course. The pathogenesis of EH remains unclear. It is considered that impaired skin barrier function, observed in several chronic skin diseases, can lead to increased susceptibility to disseminated viral infection [1]. Aside from skin disintegrity impairment, higher risk of EH is also associated with cellular and humoral immunity deficiencies. According to recent reports, high total serum IgE level, low levels of cathelicidin, as well as reduced activity of natural killer cells can contribute to widespread dissemination of viral infection [2–4]. Furthermore, increased concentration of interleukin 4, the cytokine responsible for suppressing the production of T helper cells and IFN- γ , also comprises risk factor of EH [5]. The role of immune response defects in pathogenesis of EH still remains a subject of investigation. EH occurs most often as a superimposition of Herpes simplex virus-1 infection on atopic dermatitis. Other viruses that may be involved in pathogenesis are Herpes simplex virus-2, Coxsackie A16 virus and Varicella-zoster virus. The infection is transmitted via direct contact or develops as a reactivation of the latent virus [6].

Apart from atopic dermatitis, EH has been described in patients with psoriasis, pityriasis rubra pilaris, Darier's disease, Grover's disease, Hailey-Hailey disease, autoimmune bullous dermatoses, as well as irritant and allergic contact dermatitis [7]. Furthermore, there have been reported cases of EH in patients with cutaneous T-cell lymphoma [8], or in immunocompromised patients, who had undergone laser resurfacing [9].

As atopic dermatitis typically reveals in childhood, the vast amount of patients suffering from EH is comprised of pediatric population. Immunocompromised patients are also particularly vulnerable to disseminated viral infection [6].

The clinical manifestation of EH includes an eruption of multiple painful clusters of monomorphic umbilicated vesiculopustular lesions, most commonly reported on the head, neck or upper trunk. The initial exanthema alternates into punched out ulcers covered by hematic crusts. At a later stage lesions can evolve to coalescing erosions with scalloped border [10]. Skin eruption is often associated with fever and lymphadenopathy. Most often, skin lesions subside in 2–6 weeks, without scarring [6].

In case of more severe course of the disease, infection can disseminate to the brain, liver, lungs or to the gastroin-

testinal tract. The mortality of HSV encephalitis, which is localised mainly in the temporal lobe, reaches up to 70% [11]. Involvement of periorcular region can be also associated with severe complications — patients, who suffer from herpes keratitis that occurred in the course of the disease are vulnerable to blindness [12]. Furthermore, erosions may become secondary infected with bacteria, most commonly *Staphylococcus aureus*, thus leading to bacteremia and septic shock.

The diagnosis is based mainly on clinical presentation. In case of atypical clinical course, the Tzanck smear or skin biopsy should be performed. Direct fluorescence antigen testing, another diagnostic technique, allows the precise determination of infectious agent. PCR, aside from its highest specificity and sensitivity in diagnostic approach, can be used to detect viral DNA in cerebrospinal fluid [10].

The main purposes of EH treatment are both termination of viral infection and alleviation of symptoms of the underlying disease. Antiviral therapy should be initiated as soon as possible. In less-severe disease in adults acyclovir 400 mg 5 times daily for 5 to 10 days is recommended; for pediatric patients acyclovir 25 mg/kg/day is administered in 5 equal doses for 5 to 10 days. In case of severe course of the disease, in immunocompromised patients in severe dysphagia intravenous infusion (dosing at 15 mg/kg per day) is recommended for at least 5 days. It is compulsory to adjust the dosage of the drug in patients with renal insufficiency and to recommend proper hydration in patients treated with intravenous acyclovir, due to the risk of drug precipitation as crystals in the kidneys. In case of periorcular involvement, ophthalmological care is mandatory. In the case of suspected bacterial superinfection systemic antibiotic therapy should be recommended [6]. In our patient — immunocompetent and with no history of atopic dermatitis — EH development was a consequence of disruption of epidermal barrier in the course of contact dermatitis, most probably due to occupational exposure. Immediate therapy with acyclovir combined with systemic antibiotics led to gradual resolution of skin eruption. Although EH is not a rare its development in patients with contact dermatitis has been previously described in only a few cases [13–15].

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