CLINICAL IMAGE

Ecthyma gangrenosum as the primary manifestation of acute myeloid leukemia in a previously healthy patient

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Ecthyma gangrenosum (EG) is a rare cutaneous infection that typically manifests in immunocompromised individuals and may be often associated with *Pseudomonas aeruginosa* infection.

A 50-year-old, previously healthy woman reported to the emergency department due to deterioration of a rash that had started 14 days before and fever with an onset 4 days before. At the time of presentation, the skin lesions appeared as gangrenous ulcers with black scabs surrounded by a red halo, and were localized mainly on the trunk, chest wall, and labial commissures (**FIGURE 1A–1C**). The patient had undergone a laboratory workup 6 months prior, and the results were normal.

Following the onset of fever episodes and rash deterioration, a diagnostic workup was undertaken. Laboratory investigations revealed normocytic normochromic anemia (hemoglobin, 9.0 g/dl; reference range [RR], 12.0–16.0 g/dl; mean corpuscular volume, 81 fl; RR, 80–99 fl; reticulocytes, 0.23%; RR, 0.2%–2%), leukocytosis (white blood cells [WBCs], 31.2 K/μl; RR, 3.8–10.5 K/μl; neutrophils, 19.4%; RR, 45%–75%; lymphocytes, 13.1%; RR, 20%–51%; monocytes, 67.5% atypical cells; RR, 2.0%–11.0%), and thrombocytopenia (platelets, 20 K/μl; RR, 150–450 K/μl). Peripheral blood smear showed 67% of blast cells in the WBC count, and flow cytometry was indicative of acute monoblastic / monocytic leukemia (leukemic blasts revealed CD34+, CD33+, CD13+, CD64+, CD300E+, CD4+, T1T+, CD38+, CD11a+, CD11b+, CD11+/–, CD36+/–, CD16+/–, HLADR+/–, MPO+/–, and CD14+/–). Bone marrow aspirate examination confirmed the diagnosis of acute myeloid leukemia (AML), while molecular karyotyping did not reveal any specific chromosome abnormality. A whole-body computed tomography scan was unremarkable. A skin biopsy was performed, and the histopathological examination of the lesion specimen revealed vascular necrosis with several surrounding bacteria. Gram-stained sections showed gram-negative rods surrounding the necrotic vessels, indicative of *Pseudomonas aeruginosa* infection; blood and lesion cultures confirmed the diagnosis. Following a decrease in the levels of hemoglobin and a sharp increase in inflammatory marker concentrations (C-reactive protein, 41.8 mg/dl; RR, 0–0.8 mg/dl, procalcitonin, 32.9 ng/ml; RR, <0.2 ng/ml, erythrocyte sedimentation rate, 148 mm; RR, 0–20 mm, adjusted for age, lactate dehydrogenase, 888 U/l; RR, 135–214 U/l, β2-microglobulin, 5.17 mg/l; RR, 1.42–3.21 mg/l), transfusion of red blood cells was performed, and antibiotic therapy with meropenem, linezolid, vancomycin, and isavuconazole was initiated. Despite fever deterioration, chemotherapy was introduced on day 5 of hospitalization, according to the standard schedule (idarabucin 12 mg/m^2 for 3 days and aracytin 100 mg/m^2 for 7 days). Meanwhile, the lesions evolved into necrotic ulcers involving the chest wall, oral cavity, and trunk. The patient developed sepsis and died within 3 weeks of admission to the hospital.

EG may initially present as an erythematous macule and may evolve into a hemorrhagic vesicle that turns into a gangrenous ulcer with necrotic eschar.[1] Apart from immunocompromised patients, it can also affect otherwise healthy, immunocompetent individuals, with leukemia being one of the main predisposing conditions.[2] Thus, EG occurrence in a previously healthy individual may signal undiagnosed immunodeficiency and warrants further investigation. Acute monocytic and monoblastic leukemias, occurring most commonly in young and adult people, respectively,

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FIGURE 1 Gangrenous ulcers with a gray–black eschar surrounded by an erythematous halo, localized at the trunk (**A**), the chest wall (**B**), and the labial commissures (**C**)

account for less than 5% of all AML cases. Some individuals may present with bleeding disorders, whereas cutaneous involvement is common.[2] Reports of AML cases presenting with EG as the primary manifestation are scant[3]; therefore, recognizing the underlying cause of EG is the key to a proper therapeutic approach.

ARTICLE INFORMATION

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