Journal of the Turkish Academy of Dermatology
eISSN 1307-394X

Letter To The Editor
DOI: 10.6003/jtad.1371l1

Ecthyma Gangrenosum

To the Editor.- A 58-year-old woman who presented haematology polyclinic was evaluated for high-grade fever and deterioration of her general medical condition. The patient complained of fever a necrotic lesion in her nose, and underwent physical examination. Her vital signs were: axillary temperature 39°.2C, pulse rate 108 per minute, respiratory rate 22 per minute, and blood pressure 130/70 mmHg, the findings indicated bilateral postauricular lymphadenopathy, while other system examinations were normal. On dermatological examination there were ulcerated crusty necrotic lesions with irregular and indefinite boundaries in the left edge of the nose towards the nasal openings (**Figure 1**). Blood tests showed a leucocyte count of 131000/mm^3, erythrocyte sedimentation rate of 36 mm/hr and C-reactive protein of 365 mg/dl. Urine, blood and lesional swab cultures were performed. Meropenem 3 gr/day and amikacin 1.5 gr/day was started as empirical treatment. *Pseudomonas aeruginosa* was isolated from both blood and lesional swab cultures, with the same resistance pattern. The patient, gradually decompenseted, and passed away on the fifth day of treatment. Informed consent form was taken.

The diseases associated with *Pseudomonas* septicemia were first defined by *Barker* in 1897 and were later given the name "ecthyma gangrenosum" by *Hitschmann* and *Kreibich* [**1**]. EG has a prevalence of 30% during *Pseudomonas aeruginosa* septicaemia [**2**]. Uncommonly, it may be caused by other organisms [**3**].

Ecthyma gangrenosum (EG) generally occurs in malignant diseases such as leukaemia and lymphoma, severe burns, organ transplantations, as well as patients receiving immunosuppressive treatment, and those with a chronic disease (diabetes mellitus and malnutrition). Although rare, EG may also develop in healthy individuals [**4**].

It was reported that 57% of EG cases involve the gluteal or perineal area, 30% the extremities, and the body and face in 6% of cases. As was the case in our patient, the nose may be involved [**5**]. Vascular lesions, gangrenous cellulitis, maculopapular lesions and EG are characteristic cutaneous lesions of *Pseudomonas* [**6**]. Starting as round, painless and erythematous maculae, EG lesions later develop into nodular, bullous or pustular lesions with an erythematous induration around them. The resulting typical EG lesions are brownish-black gangrenous ulcers surrounded by an erythematous halo. Almost all patients suffer from neutropenia [**1**].

A Gram stain of fluid from the central hemorrhagic pustule or bulla can facilitate rapid diagnosis. The organism can proliferate in blood, urine and tissue

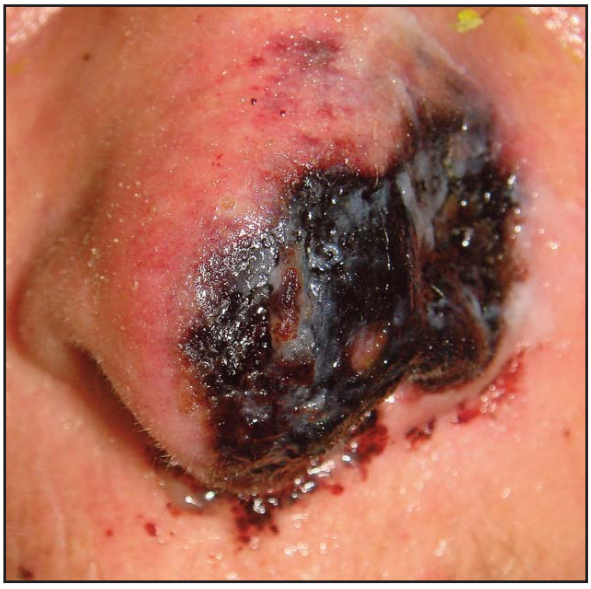

Figure 1. Necrotic lesions on the nose

Published: *J Turk Acad Dermatol* 2013; **7 (1)**: 1371l1. This article is available from: http://www.jtad.org/2013/1/jtad1371l1.pdf
Key Words: Ecthyma gangrenosum, acute myeloid leukemia, Pseudomonas aeruginosa

cultures. Histopathologically, EG is characterized by epidermal necrosis with haemorrhage and dermal infarction, usually accompanied by a mixed inflammatory cell infiltrate of lymphocytes, histiocytes and neutrophils. In general, acute mixed inflammatory cell infiltration and vascular proliferation are seen in the dermis, often involving the subcutaneous tissue. Elastases produced by Pseudomonas destroy the elastic small vessels, leading to haemorrhage and release of organisms into the surrounding tissue. Protease and endotoxin A, elaborated by bacilli, are responsible for direct tissue destruction and ulcerative lesions [**7**]. Gram staining of our patient revealed Gram-negative bacilli and *Pseudomonas aeruginosa* proliferation in both blood and ulcer cultures.

Pyoderma gangrenosum, vasculitis, cryoglobulins and septic emboli should be considered in differential diagnosis. The rate of mortality is 15% in non-bacteremic patients, compared with 38-96% in patients with bacteremia [**4**].

Early and appropriate antibiotic treatment is required to prevent fatal invasive sepsis in EG. Antibiotics used in the treatment of EG include antipseudomonal penicillins, aminoglycosides, fluoroquinolones, third-generation cephalosporins, or aztreonam. Surgical debridement of the lesion together with antibiotic treatment yields successful results [**8**]. Delay in administration of appropriate antibiotic therapy can lead to multiple lesions, resistant neutropenia and sepsis [**9**]. Our patient was started on meropenem 3 gr/day and amikacin 1.5 gr/day. The patient died on the fifth day of treatment, due to multiple organ failure resulting from sepsis.

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