

Ecthyma Gangrenosum: A dermatological manifestation of *Pseudomonas septicemia*

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Abstract

Ecthyma gangrenosum is a well recognized cutaneous manifestation of severe, invasive infection caused more often than not in immunocompromised and critically ill patients by *Pseudomonas aeruginosa*. There are isolated reports of its occurrence in normal healthy subjects and a wide range of organisms besides *Pseudomonas* have been implicated in its causation. It is important to recognize this association because of the unfavorable course allied with a delay in diagnosis and treatment.

CASE

A 9 day old term male neonate of birth weight 5 kgs, delivered via lower segment cesarean section (LSCS) presented with multiple skin lesions from the 5th post natal day. Red flat lesions initially developed over the scalp with subsequent ulceration over 3-4 days. Similar lesions developed on the back, gluteal region and both lower limbs. The skin lesions were accompanied by a continuous low-grade fever. There was no history of burns, drug intake, umbilical sepsis, catheterization or diarrhea predisposing to immuno-competent status.

On general examination an incision mark [figure1] was noted above the right tragus acquired during LSCS. Skin examination showed erythematous macules, papules, nodules, vesicles and well defined punched out gangrenous ulcers with raised erythematous borders and central black eschar over the scalp, back, gluteal regions and both lower limbs [figure2].

Figure 1

Figure 1



Figure 2

Figure 2

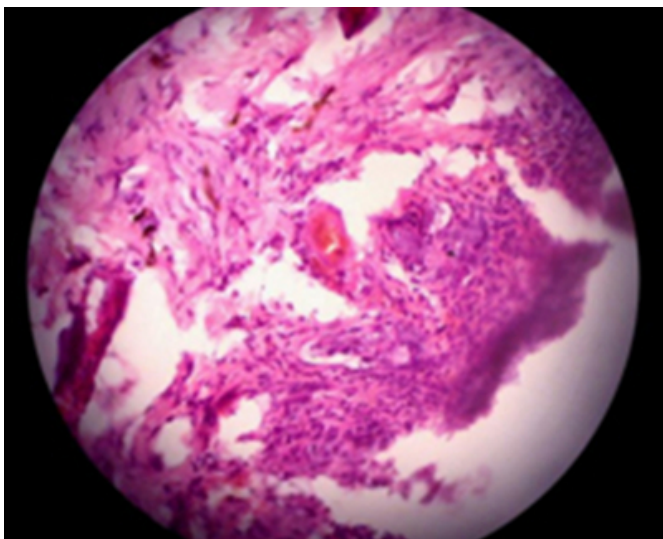


Systemic examination was unremarkable.

Investigations revealed an increased WBC count (n= 20,000) with predominant neutrophils (76%).Peripheral smear showed toxic granulations. Gram negative rods were demonstrated on gram staining of the pus. Cultures of blood, pus and biopsy grew colonies characteristic of *Pseudomonas aeruginosa* . Skin Biopsy revealed pauci immune necrotising vasculitis with hemorrhage and surrounding edema [figure3].

Figure 3

Figure 3



Chest radiography was normal .Infection by HIV was ruled out indirectly by ELISA testing of both parents. Immunoglobulin levels were normal . The diagnosis of Ecthyma Gangrenosum caused by *Pseudomonas* infection secondary to the incision acquired during LSCS was made

on clinical, bacteriological and tissue examinations.

Treatment was initiated with anti-pseudomonal antibiotics - Injection Piperacillin-Tazobactam 200mg q8hrly * 21days and injection amikacin 25mg q12hrly * 10days. Silver sulfadiazine local application and surgical debridement were also instituted. This led to resolution of the skin lesions [figure4] by 3 weeks and all the wounds healed by 6 weeks.

Figure 4

Figure 4



DISCUSSION

EG lesions characteristically begin as a painless red macules which evolve into papules and later hemorrhagic bullae which rupture to produce gangrenous ulcers with black/gray eschar surrounded by an erythematous halo [1]. The common sites of distribution of EG lesions are the gluteal or perineal region (57%), extremities (30%), trunk (6%), and face (6%) [2].

Breakdown of the mechanical defense barrier and impaired cellular or humoral immunity in combination or either one alone heighten the risk of infection by *Pseudomonas aeruginosa* [2]. The organism localizes to the vessel wall by hematogenous seeding in septicemic patients and by direct inoculation in nonbacteremic ones. Subsequent proliferation of the organism in the vessel wall produces a necrotising vasculitis by obstruction of the dermal vessels, dissolution of the elastic lamina of blood vessels by *Pseudomonas* elastase and elaboration of exotoxin A resulting in the characteristic painless, indurated ulcer with a central necrotic black eschar and surrounding erythema [3].

Ecthyma Gangrenosum typically occurs in the setting of hematological malignancies , malnutrition, severe burns, or

immunodeficiency syndromes presenting with severe neutropenia. It is important to be aware that EG can occur in absence of immunodeficiency, solely presenting as a complication of a break in the mechanical barrier of the skin or mucosa as seen in this case [2]. The absence of pain may lead to an underestimation of the serious nature of the disease which may lead to a failure in initiation of treatment [4]. EG requires prompt diagnosis as early institution of antipseudomonal agents reduces the high mortality associated with pseudomonal sepsis [3].

Treatment should begin on clinical grounds not waiting for laboratory confirmation with an, an antipseudomonal penicillin (piperacillin) in conjunction with an aminoglycoside (gentamicin). Combination therapy is far superior to monotherapy [5].

In conclusion physicians must be aware of the association between EG and Pseudomonas septicemia in both

immunocompromised and healthy subjects, to initiate prompt antipseudomonal therapy for improved outcomes.

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