

Case Report

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Ecthyma gangrenosum in a neonate

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ABSTRACT

Ecthyma gangrenosum (EG) is a rare skin infection caused by *pseudomonas aeruginosa*. It typically occurs in immunocompromised and critically ill patients but can occur rarely in healthy children. 28 days old neonate presented with widespread vesiculobullous lesions which ruptured to form punched out gangrenous ulcers with black eschar. Blood and pus culture grew *Pseudomonas aeruginosa* and diagnosis of EG due to *pseudomonas* was made. Despite appropriate measures, baby succumbed to the illness. Rapid progression of the lesions and high mortality rate emphasize the importance of early suspicion and proper treatment.

Keywords: Ecthyma gangrenosum, Neonate, *Pseudomonas*

INTRODUCTION

Ecthyma gangrenosum (EG) is a cutaneous manifestation of invasive infection usually caused by *pseudomonas*, but can be caused by many bacteria, fungal and viral infections. The lesions characteristically appear as small indurated papulo-vesicles progressing rapidly to necrotic ulcers with surrounding erythema and a central black eschar. It is common in immunocompromised patients, especially those who are neutropenic and critically ill patients but can occur rarely in healthy individuals.¹⁻³ EG has also been described in infants and young children with transient risk factors, such as concurrent viral infection and recent antibiotic therapy.⁴ It has been proposed that such factors may disrupt normal host defences by weakening the mucosal barrier of the gastrointestinal tract or temporarily affecting neutrophil number and/or function.⁵

Common sites are the gluteal or perineal region (57%) extremities (30%) trunk (6%) and face (6%). Fever and other constitutional symptoms may be present depending on the extent of the underlying infection and the patient's immune status.⁶

CASE REPORT

A 28 days old female baby, presented with multiple fluid filled lesions for 5 days' duration. The lesions initially appeared over the upper extremity and extended to involve back, gluteal, scalp and both lower limbs over next two days. It was associated with poor feeding for two days. There was no history of accompanying fever, vomiting or convulsions. Baby was born at term normal vaginal delivery with low birth weight, 2.3 kg. She was a product of non-consanguineous marriage. There was history of NICU admission for 3 days for perinatal asphyxia. Baby was discharged on day 5 of life on exclusive breast feeds. Mother started cow's milk on day 10 of life. Baby had received birth dose of vaccination.

On examination baby was sick looking, septic with shock. Anthropometry showed weight - 2.5 kg, length- 48 cm. There was pallor, without any cyanosis and icterus. Multiple large well defined vesicular and bullous lesion with erythematous borders noted predominantly over the extremities (Figure 1, 2). The lesions ruptured to form punched out gangrenous ulcer with depressed necrotic black eschar, crusted centre and raised edges (Figure 3). Scalp was severely affected

exposing the underlying bone. Systemic examination was normal (Figure 4).



Figure 1: Bullous lesions with erythematous borders.



Figure 2: Skin lesions predominantly over the extremities.



Figure 3: Gangrenous ulcers with necrotic black eschar.



Figure 4: Scalp is also involved.

Initially a diagnosis of neonatal septicaemia with gangrene was made. Baby was treated with supportive measures, inotropes for shock. Parenteral antibiotics piperacillin with tazobactam, amikacin and metronidazole were started. Silver sulfadiazine local application was also instituted. Investigations revealed leukopenia ($n = 2100$) with thrombocytopenia (50,000). Peripheral smear showed toxic granulations. Chest x-ray was normal. Blood and pus cultures grew *Pseudomonas aeruginosa*. The diagnosis of ecthyma gangrenosum due to *pseudomonas* infection was made. Immunoglobulin profile and HIV ELISA were normal. Despite above measures, baby succumbed to the illness on day 3 of hospitalization.

DISCUSSION

There have been few case reports of EG in neonates especially in preterm babies. These babies at risk due to the parenteral nutrition and prolonged hospital stay. Though our case was low birth weight with faulty feeding practice and failing to thrive, the exact cause of infection could not be identified. There was no underlying immunodeficiency as evaluated by immunoglobulin profile and HIV ELISA. EG requires prompt diagnosis because delay in institution of anti-*pseudomonas* agents is associated with high mortality. Even with appropriate therapy the mortality for *Pseudomonas septicemia* in the immunocompromised remains high, ranging from 38-77%, with septic shock and multisystem organ failure commonly occurring. In these case prognosis for patients with the non-septicemic variant of EG is much better, with a reported mortality rate of 15%.⁷

CONCLUSION

Although EG classically occurs in immunocompromised patients, the same entity may arise in otherwise healthy children. As the appearance of EG can be highly variable, it should always be considered in the differential

diagnosis for septic patients presenting with neutropenia and a new skin lesion. Poor prognosis is associated multiple lesions, delay in treatment and neutropenia. Rapid progression of the lesions and high mortality rate emphasize the importance of early suspicion and proper treatment

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REFERENCES

1. Huminer D, Igra SY, Morduchowicz G. Ecthyma gangrenosum without bacteremia. Report of six cases and review of the literature. *Arch Intern Med.* 1987;2013:299-301.
2. Foca MD. *Pseudomonas aeruginosa* infections in the neonatal intensive care unit. *Semin Perinatol.* 2002;2013:332-9.
3. Patel JK, Perez OA, Viera MH. Ecthyma gangrenosum caused by *Escherichia coli* bacteremia: a case report and review of the literature. *Cutis.* 2009;2013:261-7.
4. Koo SH, Lee JH, Shin H, Lee JI. Ecthyma gangrenosum in a previously healthy infant. *Arch Plast Sur.* 2012;39:673-5.
5. Pechter PM, Marchione R, Milikowski C, Berman B. Ecthyma gangrenosum secondary to *Staphylococcus aureus* in an infant with transient neutropenia. *Pediatr Dermatol.* 2012;29:320-3.
6. Fink M, Conrad D, Matthews M, Browning JC. Primary ecthyma gangrenosum as a presenting sign in a child. *Dermatol Online J.* 2012;18:3.
7. Pena C, Suarez C, Gozalo M, Murillas J, Almirante B. Prospective multicenter study of the impact of carbapenem resistance on mortality in *Pseudomonas aeruginosa* bloodstream infections. *Antimicrobial Agents and Chemotherapy.* 2011;56:1265-72.

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