Case Report

Ecthyma Gangrenosum in a Preterm Neonate with Early-Onset Sepsis: A Case Report

Janu Chhetri[1], Muhammad Asif Siddiqui[2], Inisha Sapkota[1], Naveed Akbar Hotiana[3]

[1]MBBS student, Fatima Jinnah Medical University, Lahore, Pakistan; [2]Department of Neonatology, Fatima Jinnah Medical University, Lahore, Pakistan; [3]Department of Pediatrics, Fatima Jinnah Medical University, Lahore.

Correspondence to: Muhammad Asif Siddiqui, Email: drmasirmc@gmail.com

ABSTRACT

Ecthyma gangrenosum (EG) is a rare but fatal condition in preterm babies with characteristic skin manifestations primarily associated with critically ill and immunocompromised states, often caused by Pseudomonas aeruginosa (Pa) infection but also resulting from various other bacterial and fungal pathogens. This case report describes ecthyma gangrenosum in a preterm (31 weeks), low-birth-weight (1.8 kg) neonate with early-onset neonatal sepsis (EONS) born to a primigravida mother with prolonged premature rupture of membranes (PROM). On the second day of life, multiple erythematous cutaneous lesions appeared and rapidly evolved into characteristic necrotic ulcers, accompanied by systemic manifestations of sepsis. Diagnostic challenges included the overlap of EG symptoms with other neonatal skin conditions and the absence of positive culture results. Despite these challenges, prompt recognition and initiation of broad-spectrum antibacterial therapy, supportive measures, and multidisciplinary care resulted in clinical improvement with eventual discharge on oral linezolid on the 17th day in healthy condition. This case highlights the importance of early recognition of EG in neonates, necessitating early initiation of appropriate therapy and close monitoring for improved outcomes in this vulnerable population. Further research is needed to enhance understanding and optimize management strategies for EG in neonatal sepsis.

Keywords: Ecthyma Gangrenosum; Sepsis; Infant; Premature; Low Birth Weight; Premature Rupture of Fetal Membranes

INTRODUCTION

Ecthyma gangrenosum (EG) is rare and is due to *Pseudomonas aeruginosa*, although other bacteria, viruses, and fungi usually in immunocompromised individuals; it can also occur in neonates and children without any underlying immune disorder, and it is associated with characteristic skin manifestations often observed in immunocompromised individuals, typically signifying severe sepsis, frequently linked to *Pseudomonas aeruginosa* (Pa).[1,2] The lesions typically present as gangrenous ulcers with erythematous borders and often feature raised, purplish, indurated, and rolled edges. Additionally, erythematous, violaceous, elevated, and indurated nodules may appear with or without fluctuation, alongside other non-specific lesions like vesicles, small papules, and cellulitis.[3] EG primarily affects the axillary and anogenital areas but can also involve the arms, legs, trunk, and face. The classic appearance results from perivascular invasion and subsequent ischemic necrosis of the skin.[4] Early identification is very crucial, and initiation of empiric broad-spectrum antibacterial therapy is crucial for effective treatment; without early recognition and treatment, it is associated with poor prognosis and mortality.[5,3] This case describes the development of ecthyma gangrenosum in a preterm, low-birth-weight neonate with early-onset neonatal sepsis. It highlights the diagnostic challenges of culture-negative disease and emphasizes the importance of early clinical recognition, prompt empirical antimicrobial therapy, and multidisciplinary management to achieve a favorable outcome.

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CASE PRESENTATION

A preterm female baby was delivered at 31 weeks gestation to a primigravida mother who had a history of per vaginal leakage for one week prior to delivery and presented with premature rupture of membranes (PROM). The baby was delivered via emergency lower segment cesarean section (LSCS) in the Department of Obstetrics and Gynecology, Sir Ganga Ram Hospital (SGRH), Lahore, a tertiary care teaching hospital. The newborn had a spontaneous cry at birth, indicating initial stability with a weight of 1.8 kg. However, after two

hours, she developed rapid breathing and signs of distress, including nasal flaring and chest retractions. She was transferred to the Pediatric Intensive Care Unit (PICU), Sir Ganga Ram Hospital for closer monitoring. On examination, her respiratory rate was found to be 66/min, temperature of 37.6°C, heart rate of 158 beats/min, blood pressure of 50/30 mmHg, capillary refill time of 5 sec, along with diminished neonatal reflexes and an inability to accept feeds. The remainder of the systemic examination was unremarkable. Diagnosis of early onset neonatal sepsis (EONS) was made. Laboratory investigations revealed a total leukocyte count of 27,000/µl, an absolute neutrophil count of 23,200/µl, and a CRP level of 12 mg/dl. Chest X-ray showed bilateral infiltrates. After fluid resuscitation with normal saline, first-line antibiotics IV cefotaxime and amikacin were started for broad-spectrum coverage. Supportive therapy included temperature maintenance, oxygen therapy, vitamin K, maintenance IV fluids, and nasogastric feeding. Blood cultures were sent for microbiological evaluation.

On the second day of life, multiple firm, erythematous lesions of varying sizes (average 2×2 cm) developed on the arms, chest, and legs (Figure 1). The newborn remained persistently tachypneic with fever and hypotension. Ecthyma gangrenosum (EG) with probable Pseudomonas septicemia and disseminated intravascular coagulation (DIC) was suspected. Antibiotic therapy was adjusted based on clinical suspicion, with the addition of IV ceftazidime and piperacillin-tazobactam because cefotaxime is not for Pseudomonas infection. Over the next five days, the skin lesions became more necrotic and spread further. A gangrenous ulcer with a gray-black eschar surrounded by an erythematous halo developed on the right cheek, accompanied by hemorrhagic bullae on the trunk. Pallor and mild abdominal distension were noted. The baby was continuously oxygen-dependent, requiring supplemental oxygen to maintain adequate oxygen saturation levels. She was vigorously managed with antibiotic therapy, maintenance IV fluids, dobutamine infusion for shock, vitamin K, calcium gluconate, $MgSO_4$, and nasogastric feeding. Labs were repeated, and close monitoring was conducted. She also required transfusions of packed red blood cells and fresh frozen plasma. When her condition did not improve and the culture report was found to be negative, on the 8th day, the antibiotic regimen was changed to IV meropenem and linezolid; as the culture was negative, there was clinical deterioration. Fusidic acid was applied over the lesion. The treatment continued with close monitoring, and labs were repeated. She progressively showed improvement over the next few days. Pallor and abdominal distension showed gradual improvement. Skin biopsy could not be performed. On the 15th day of hospitalization, the neonate's vitals stabilized, with a respiratory rate of 46/min and heart rate of 142 beats/min. The skin lesions had regressed to mildly erythematous patches, and the cheek ulcer had healed (Figure 2). She was weaned off oxygen and was observed for any discomfort. The neonate was discharged on the 17th day, stable, oxygen-free, and oral feeding was well-tolerated. She showed no signs of distress and was vitally stable. The discharge plan included oral linezolid with instructions for close monitoring of danger signs. Now the baby is perfectly fine and in good health.

Figure 1: Multiple erythematous and necrotic lesions on the neonate's chest and limbs, characteristic of ecthyma gangrenosum, before initiation of targeted therapy.

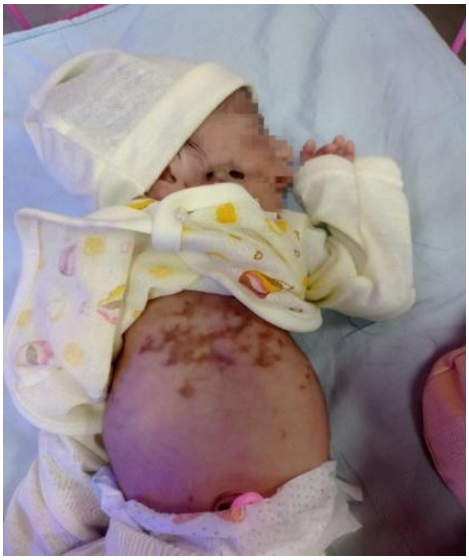

Figure 2: Resolution of necrotic lesions with residual erythema and scarring following 17 days of antibiotic therapy and supportive care.

DISCUSSION

Ecthyma gangrenosum (EG) is usually considered as a disease of profoundly immunocompromised hosts, and the recent paediatric literature shows as: reports from 2021–2024 describe EG in a 5-month-old with septic shock, a 7-year-old with XDR *Acinetobacter baumannii*–Aspergillus co-infection, and a previously healthy 16-month-old with fulminant Staphylococcus aureus infection, this means it can occur in relatively healthier population as well as compared to preterm.[6-8] However, preterm babies are immunocompromised, as in our case, a preterm neonate with early-onset sepsis (EOS), since a premature infant has not yet completed third-trimester transplacental IgG transfer, had an immature neutrophil pool, and typically acquires infection by an ascending route through ruptured membranes rather than nosocomial exposure. This is how our case is different from the paediatric population.

Although EG is rare in preterm babies as well, there are a few case reports from the literature showing that Basu and Kumar described a premature, low-birth-weight neonate born to a mother with chorioamnionitis, already on antibiotics for presumed EOS, who developed gangrenous ulcers and died with *Pseudomonas aeruginosa* on blood culture susceptible only to imipenem.[9] Serrano-Martín and colleagues reported facial EG in two preterm twins, noting that lesions can precede overt sepsis by days, making cutaneous recognition rather than culture the practical trigger for treatment.[10] Pathak and colleagues documented the first EG case caused by invasive E. coli in a 34-week neonate [12], Ward Platt and colleagues reported EG in a preterm neonate in intensive care[14], Sahoo and colleagues described Pseudomonas bacteremia with EG in a 4-day-old term neonate[13], and, most recently, Lalaoui and colleagues reported a 34+5-week preterm twin, initially treated for presumed EOS, in whom skin culture grew *Klebsiella pneumonia*e and *Enterococcus faecium* and blood culture grew *Enterococcus faecium* alone, in an infant explicitly described as immunocompetent.[11] Together, these reports show that EG in preterm neonates is not confined to Pseudomonas or an immature immune system; rather, it is associated with many other organisms with poor outcomes and mortality and relying on cultures and sensitivity may lead to death and poor outcomes. So, early clinical recognition is essential.[9] The unique thing about our case report is “EG in a preterm neonate," with a more specific combination: a clearly documented obstetric chain of premature rupture of membranes, maternal infection, prolonged labor and birth asphyxia; blood cultures that remained persistently negative throughout despite an unmistakable EG phenotype; and survival to discharge, in contrast to the fatal outcome in the closest comparable case. [9]

Persistently negative blood cultures do not, on their own, argue against the diagnosis. Gençer and colleagues reported a previously healthy man whose blood cultures stayed sterile while culture of the biopsy material itself grew *Pseudomonas aeruginosa*, concluding that EG should remain on the differential even with negative cultures because bacteremia is not required for the cutaneous lesion to develop.[15] This is consistent with EG being fundamentally a clinicopathologic diagnosis, made from the lesion's characteristic evolution from macule to hemorrhagic bulla to necrotic ulcer with an indurated rim, ideally supported by tissue-level confirmation; Gonzaga and colleagues showed in neutropenic adults that direct microscopy of an early skin biopsy identified the causative organism the same day, before cultures returned, and changed empiric therapy accordingly. In this neonate, prior antibiotic exposure for presumed EOS before the skin findings evolved is a possible explanation for culture sterilization, mirroring the Basu and Kumar and Lalaoui cases[9,11]; our limitation was the inability to perform a skin biopsy test apart from cultures, which was done by many other case reports to support diagnosis as the appropriate step once cultures are unhelpful.[15]

The sequence of antibiotic changes also deserves a mechanistic explanation rather than a bare chronology. Empiric EOS coverage is broadened toward anti-pseudomonal agents only once a necrotic lesion raises suspicion of EG, which explains the need for escalation here, as in the Basu and Kumar and Lalaoui cases, both of which likewise began with standard EOS coverage before the skin findings prompted a change.[9,11] Biofilm formation by Pseudomonas and related organisms, which shields bacteria from antibiotic penetration even when isolates remain susceptible on standard testing, offers a more specific explanation for a slow, non-linear clinical response than invoking resistance in the abstract. The eventual step-down to oral linezolid at discharge is itself informative, since linezolid has no Gram-negative activity; its use implies the working diagnosis had shifted toward a resistant Gram-positive organism, aligning this case more closely with the non-pseudomonal preterm report by Lalaoui and colleagues than with the classically pseudomonal preterm cases.[9,10,12] Similarly, immunoglobulin therapy is better explained here through a specific mechanism than as a generic option for "combined immunodeficiency": preterm infants have physiologically low IgG because transplacental transfer occurs mainly in the third trimester, and that gap, rather

than a broad diagnostic category, is the actual rationale for considering IVIG as an adjunct in this population.

Against this small existing case series, the present case's contribution is narrow but real: it is the only one with a clearly documented ascending-infection antecedent acting together with EOS, the only one with cultures that remained negative throughout despite a fully evolved EG lesion, and, in contrast to the only other reported case of EG complicating EOS in a premature infant, a survivor.[9] The absence of a confirmatory skin biopsy and the unidentified causative organism are real limitations, and they point to what future reports in this population should do differently: obtain skin biopsy with Gram stain and tissue culture before antibiotic escalation, so the organism-level question this case leaves open can be answered in the next one.

CONCLUSIONS

Ecthyma gangrenosum should be considered in preterm neonates with early-onset sepsis who develop rapidly progressive necrotic skin lesions, even in the absence of microbiological confirmation. This case highlights the importance of high index of suspicion, early clinical recognition, prompt initiation of empirical broad-spectrum antimicrobial therapy, and multidisciplinary supportive care in improving clinical outcomes. Increased awareness of this rare but potentially life-threatening presentation may facilitate timely diagnosis and appropriate management in similar neonatal cases.

Author Contributions

JC: Conception and design, analysis and interpretation of data, drafting the article, critical revision for important intellectual content, final approval.
MAS: Conception and design, analysis and interpretation of data.
IS: Analysis and interpretation of data, drafting the article.
NAH: Acquisition of data, conception and design, analysis and interpretation. All authors read and approved the final copy of the manuscript.

Artificial Intelligence (AI) Use Disclosure

ChatGPT (OpenAI) was used during the preparation of this manuscript for language editing and improvement of manuscript clarity. The AI tool was not used to generate or manipulate original research data, perform statistical analysis, or independently develop scientific conclusions or interpretations. All AI-assisted text and suggestions were critically reviewed, edited, and approved by the authors. The authors remain fully responsible for the accuracy, originality, integrity, scientific content, data interpretation, and conclusions of the manuscript.

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