Neonatal purpura fulminans caused by *Acinetobacter baumannii*: Unusual occurrence of three coincident cases

Sir,

Neonatal purpura fulminans is a potentially lethal disorder, characterized by disseminated intravascular coagulation triggering dermal thrombosis, which leads to progressive hemorrhagic necrosis of the skin in the newborn.[1] Neonatal purpura fulminans may be congenital, caused by protein C and protein S deficiency or acquired, resulting from septicemia caused by Group B *Streptococcus* and Gram-negative organisms.

Three full-term neonates, born within 12 hours in a tertiary care facility, developed multiple hemorrhagic blisters and ecchymosis over acral areas and thighs within 12–36 hours of birth. All three were born healthy with a normal Apgar score. There was no family history of coagulation disorders.The third neonate was born to consanguineous parents.

In the first neonate, the acral bullous lesions turned necrotic and hemorrhagic within a day with evident gangrenous changes by day three. These lesions rapidly progressed to involve thighs and toes [Figure 1] and were associated with systemic features of neonatal sepsis like lethargy, fever, tachycardia and poor feeding. By day five, he worsened and developed acute respiratory distress. The other two neonates had a relatively less fulminant progression, with acral hemorrhagic bullae turning necrotic [Figures 2 and 3] within 24 hours followed by the development of gangrene. No new lesions appeared after 72 hours of disease onset. Clinical features and clustering of cases led to the diagnosis of neonatal purpura fulminans secondary to sepsis The differential diagnoses of meningococcemia/septic vasculitis and thrombotic thrombocytopenic purpura were also considered. Relevant investigations were undertaken summarized in Table 1. All neonates were managed in the neonatal intensive care unit. In addition to the general care and intravenous fluids (as per clinical assessment), all the neonates were administered parenteral antibiotics and fresh frozen plasma. While the first neonate succumbed to septicemia by day seven, the other two neonates showed progressive improvement with healing of skin lesions. The course of their stay in the hospital and the treatment offered have been tabulated [Table 2]. Multiple swabs from the concerned health care workers, instruments and surfaces of the labor room failed to reveal any pathogens. Furthermore, detailed evaluation (history and clinical examination) of the mothers failed to reveal any focus of infection. The labor room was disinfected as per hospital infection control committee guidelines and no further cases were reported.

Neonatal purpura fulminans generally presents 2-12 hours after birth as ecchymotic lesions over perineum, thighs and abdomen that turn rapidly into purpuric papules, bullae and hemorrhagic necrosis, the latter being the hallmark of the condition.[2] Acquired neonatal purpura fulminans is mainly due to severe infections with *Streptococcus*, *Escherichia coli*, varicella, meningococcus and pneumococcus. Recently, *Klebsiella oxytoca* and *Citrobacter* have been reported to cause neonatal purpura fulminans.[3] We report here *Acinetobacter baumannii,* a Gram-negative coccobacillus, as the causative organism for

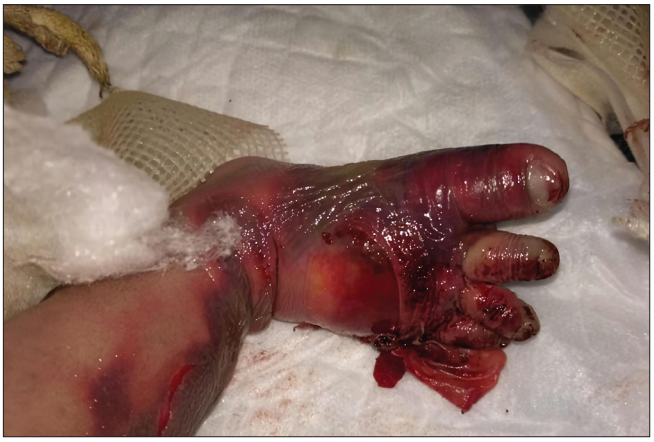

Figure 1: First neonate with acral lesions at day two

How to cite this article: Singal A, Dhir B. Neonatal purpura fulminans caused by *Acinetobacter baumannii*: Unusual occurrence of three coincident cases. Indian J Dermatol Venereol Leprol 2022;88:132.

Received: October, 2020 **Accepted:** June, 2021 **EPub Ahead of Print:** August, 2021 **Published:** December, 2021

DOI: 10.25259/IJDVL_1273_20 **PMID:** 34491670

Table 1: Summary of investigations

Investigations	Neonate 1	Neonate 2	Neonate 3
TLC† (10^9/L)	26	15.8	32.1
Platelet count (109/L)	440	354	420
FDP (mg/L)‡	5-20	5-20	5-20
d-dimer (ng/L) normal range (<250 ng/mL)	1943	783→481	2249→1715
PT/ APTT/INR§ (s)	12/33/1.0	12/26/1.1	11/29/1.0
Protein C *and* S levels	WNL	WNL	WNL
CRP (mg/L)¶ Range	>0.6	<0.6	<0.6
Blood culture and sensitivity	*Acinetobacter baumannii*; sensitive to meropenem, vancomycin, piperacillin	*Acinetobacter baumannii;* sensitive to ampicillin, amoxycillin/clavulanic acid, gentamycin, vancomycin, colistin	*Acinetobacter baumannii*; sensitive to meropenem, vancomycin, teicoplanin
Pus culture and sensitivity	*Acinetobacter baumannii;* sensitive to cefepime, meropenem, vancomycin, trimethoprim/sulfamethoxazole	No growth	*Acinetobacter baumannii*; sensitive to cefepime, meropenem, vancomycin, ciprofloxacin
Necrosis progression	+++	++	++
New lesions after a week	+++	No lesions	No lesions

†TLC: Total leukocyte count, WNL: Within normal limits, N.A: Not available, ‡FDP: Fibrin degradation product, ¶CRP: C-reactive protein, §PT/APTT: Prothrombin time/activated prothrombin time. INR-International Normalised Ratio

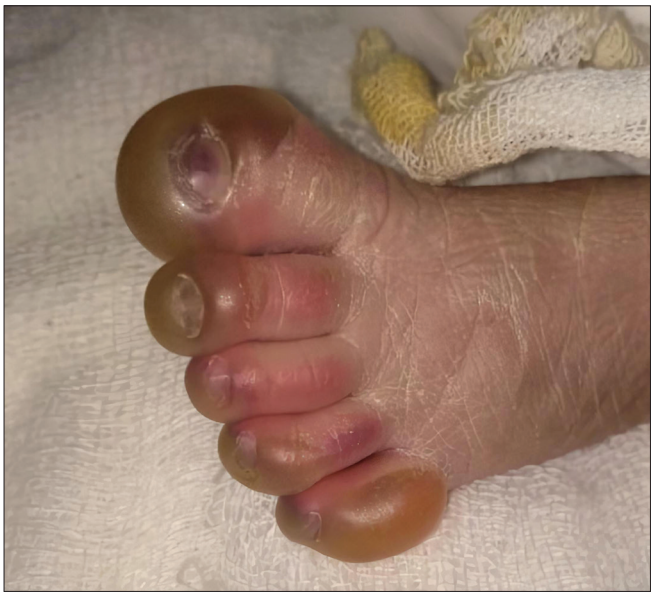

Figure 2: Second neonate with acral blisters at day two

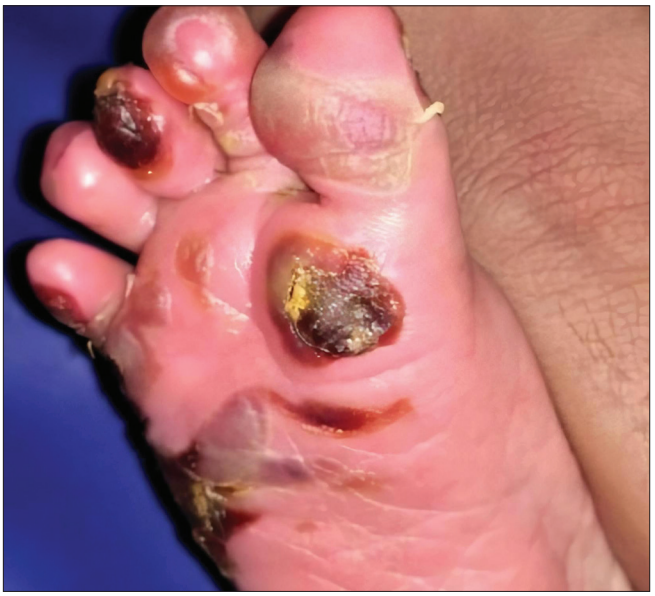

Figure 3: Third neonate with acral blisters and purpura at day five

neonatal purpura fulminans. It accounts for 2-10% of Gram-negative hospital infections. It has been labeled by Infectious Diseases Society of America as one of the six most important multidrug-resistant microorganisms in hospitals worldwide.[4] Even though *Acinetobacter* infections are more frequent in immunocompromised individuals, no such immunosuppressed state was noted in any of the neonates.

During the acute phase of neonatal purpura fulminans, laboratory findings are of disseminated intravascular coagulation including thrombocytopenia, increased fibrin degradation products, d-dimer, prolonged prothrombin time and activated partial thromboplastin time. Proteins C and S levels may be reduced in both acquired and inherited cases. In such a scenario, testing of parents is recommended. Confirmatory testing at three and six months of age is vital in neonates.

In the management of neonatal purpura fulminans, the goal is to maintain platelet count above 50,000/mm^3 and fibrinogen levels less than 1 g/L. Early empiric broad-spectrum antibiotic therapy covering the reported pathogens is paramount. It may be modified based on the sensitivity of isolates. Fresh frozen plasma (at a dose of 10–20 mL/kg every 6–12 hours) has a critical role in replacing the depleted levels of proteins C and S and preventing the progression of the disease.[5] With strict aseptic precautions followed in obstetric and neonatal care, neonatal purpura fulminans is becoming a rare entity. However, a high

Table 2: Course during hospital stay and treatment

Neonate 1	Neonate 2	Neonate 3
Inj. meropenem + Inj. vancomycin†	Inj. amoxicillin + clavulanic acid and inj. amikacin†	Inj. meropenem + Inj. vancomycin†
↓	↓	↓
three units FFP‡ over three days	one unit FFP on day two	three units FFP on day two
↓	↓	↓
Worsened by day five	Clinical improvement on day four	TLC reduced to $13\times10^{9/L}$ (from 32)
Intubated and put on mechanical ventilation	↓	↓
↓	Significant improvement by day seven	Clinical improvement by day four/five
Further deterioration and patient expired on day seven		

†As per weight, ‡FFP: Fresh frozen plasma; TLC_Total Leukocyte Count

index of suspicion for neonatal purpura fulminans should be kept for newborns presenting with hemorrhagic cutaneous necrosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Archana Singal, Bhawuk Dhir

Department of Dermatology and STD, University College of Medical Sciences and GTB Hospital, University of Delhi, Delhi, India

Corresponding author:
Dr. (Prof.) Archana Singal,
Department of Dermatology and STD, University College of Medical Sciences and GTB Hospital, University of Delhi, Delhi, India.
archanasingal@hotmail.com

References

1. Price VE, Ledingham DL, Krümpel A, Chan AK. Diagnosis and management of neonatal purpura fulminans. Semin Fetal Neonatal Med 2011;16:318-22.
2. Verma P, Pandhi D, Yadav P, Dhawan AK. Neonatal purpura fulminans due to methicillin resistant *Staphylococcus aureus*. Pediatr Dermatol 2013;30:266-7.
3. Disse SC, Meyer S, Baghai-Arassi A. Sepsis-associated purpura fulminans due to *Klebsiella oxytoca*. Dtsch Arztebl Int 2018;115:784.
4. Antunes LCS, Visca P, Towner KJ. Acinetobacter baumannii: Evolution of a global pathogen. Pathog Dis 2014;71:292-301.
5. Mazzone L, Schiestl C. Management of septic skin necroses. Eur J Pediatr Surg 2013;23:349-58.