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Severe community-associated methicillin-resistant *Staphylococcus Aureus* necrotizing pneumonia in early infancy: a case report

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ABSTRACT:

- ***Background:*** Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) is an emerging cause of severe infections in previously healthy children, including rare but life-threatening necrotizing pneumonia.
- ***Case Report:*** We report the case of a previously healthy 2-month-old male infant who developed necrotizing pneumonia and empyema secondary to CA-MRSA. The clinical course required prolonged intravenous targeted antibiotic therapy and pleural drainage during an extended hospital stay. A household decolonization protocol was implemented upon discharge to prevent recurrence.
- ***Conclusions:*** This case highlights the capacity of CA-MRSA to cause invasive, severe disease in young infants, even in the absence of classic risk factors. Early recognition of potential MRSA-related infections, timely diagnostic imaging, and prompt initiation of appropriate antimicrobial therapy are critical for improving outcomes.
- ***Keywords:*** Community-acquired MRSA, Necrotizing pneumonia, Empyema.

INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA), historically linked to healthcare environments, has increasingly established itself as an important cause of community-acquired infections in pediatric populations[1]. Over the past two decades, community-acquired MRSA (CA-MRSA) has shown a rising incidence among infants and young children, with several regions reporting an increasing proportion of MRSA among *Staphylococcus aureus* infections in otherwise healthy pediatric patients[1,2].

The pathogenicity of CA-MRSA is multifactorial. While Panton–Valentine leukocidin (PVL) is often associated with extensive tissue necrosis, other virulence determinants, including α-toxin-mediated epithelial injury and immune-evasion mechanisms, also contribute to severe invasive disease[2,3].

This case illustrates severe necrotizing pneumonia caused by a PVL-negative CA-MRSA strain in an otherwise healthy infant. Most published pediatric cases[3] of CA-MRSA necrotizing pneumonia involve PVL-positive strains, and the absence of this toxin in our patient highlights that significant pulmonary destruction can occur even without PVL expression, emphasizing that PVL status should not be used in isolation to gauge disease severity.

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CASE PRESENTATION

A previously healthy 2-month-old male infant presented to the emergency department with fever, dry cough, and intermittent grunting that began earlier on the day of admission. The fever occurred every 6 hours, with a maximum recorded temperature of 38°C. Despite these symptoms, the infant maintained adequate feeding (exclusive breastfeeding), as well as bowel movements and urination. There were no reports of vomiting, rash, or other systemic symptoms. The infant resided in a rural household with family members and a domestic dog. There was no recent travel history or known sick contacts.

Prenatal and perinatal history were unremarkable. Growth and neurodevelopment were normal, with no significant family history of hereditary or infectious diseases. One week prior to admission, he had developed erythema and bullous, exudative lesions in the perineal area. A swab was taken from the exudate, and the lesion was initially treated with topical fusidic acid. MRSA was later isolated from the lesion culture.

Initial examination revealed a well-appearing infant without respiratory distress or hypoxemia. Chest auscultation demonstrated normal bilateral breath sounds. Laboratory tests showed leukocytosis (27,100/µL) with neutrophilia (20,800/µL), elevated C-reactive protein (123.3 mg/L), and procalcitonin level of 0.25 ng/mL. Urinalysis was unremarkable. A blood culture was obtained. Chest radiograph revealed extensive right-sided multilobar pneumonia (Figure 1). Respiratory viral testing, including SARS-CoV-2, Influenza A and B, and respiratory syncytial virus, was negative. Lumbar puncture revealed 15 white blood cells/mm^3 (2 red blood cells/mm^3), normal glucose (65 mg/dL), and a protein concentration of 0.44 g/L. Empirical intravenous antibiotics - ampicillin (400 mg/kg/day, every 6 hours) and cefotaxime (300 mg/kg/day, every 8 hours) - were initiated according to the institutional protocol, considering pneumonia with presumed sepsis and possible meningitis. On admission, a nasal swab for MRSA screening tested negative by both polymerase chain reaction (PCR) and standard culture.

Despite initial treatment, the infant's condition deteriorated on day 2, becoming lethargic, with reduced oral intake. Examination revealed tachycardia, tachypnea, and intercostal retractions. Pulmonary auscultation demonstrated markedly diminished breath sounds over the right hemithorax. Laboratory studies showed worsening leukocytosis (29,300/µL; neutrophils 21,600/µL), a markedly elevated C-reactive protein level (321.6 mg/L), and a procalcitonin level of 7.9 ng/mL. Due to hypoxemia, supplemental oxygen by nasal cannula at 1 L/minute was initiated.

Thoracic ultrasound revealed extensive involvement of the right lung with features of necrotizing pneumonia and a complex ipsilateral pleural effusion (Figure 2).

Contrast-enhanced chest computed tomography (CT) confirmed right lung necrotizing pneumonia with multiple pneumatoceles and moderate pleural effusion causing compressive atelectasis (Figure 3).

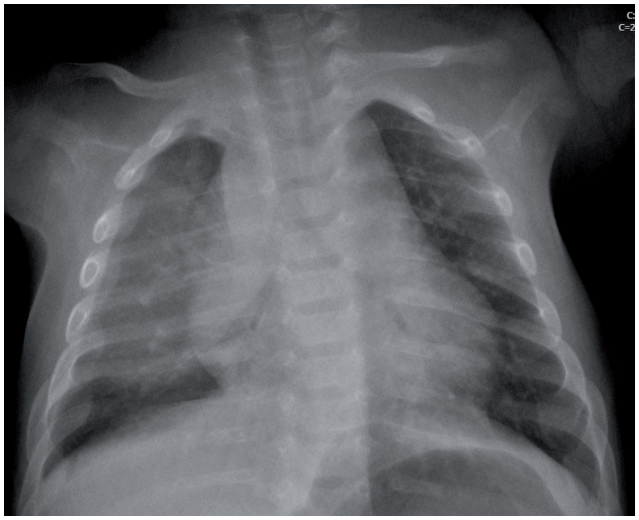

Figure 1. Admission anteroposterior chest X-ray reveals an area of increased opacity in the lower half of the right lung, consistent with parenchymal consolidation.

Given the rapid clinical deterioration and radiological evidence of necrotizing pneumonia, MRSA infection was strongly suspected on day 2, prompting therapy escalation. Vancomycin (60 mg/kg/day every 6 hours) and clindamycin (40 mg/kg/day every 6 hours) were initiated (Figure 4). Ampicillin was discontinued the same day. Cefotaxime was temporarily maintained to ensure meningitis coverage until cerebrospinal fluid cultures (CSF) results were negative; it was then discontinued, completing a 10-day course. A chest tube was placed, draining

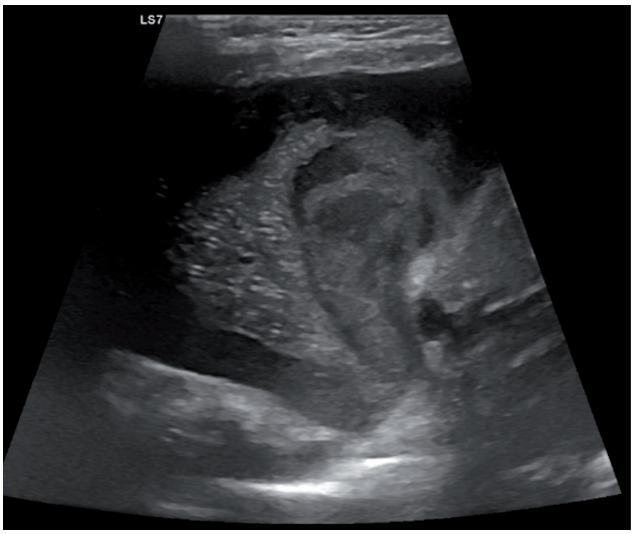

Figure 2. Day-2 thoracic ultrasound revealed extensive consolidation of the right hemithorax, with hypoechoic and heterogeneous areas, consistent with necrotizing pneumonia. An ipsilateral pleural effusion measuring up to 26 mm in thickness was also identified, containing internal echogenic debris and septations, findings consistent with a complex, likely infected exudative effusion.

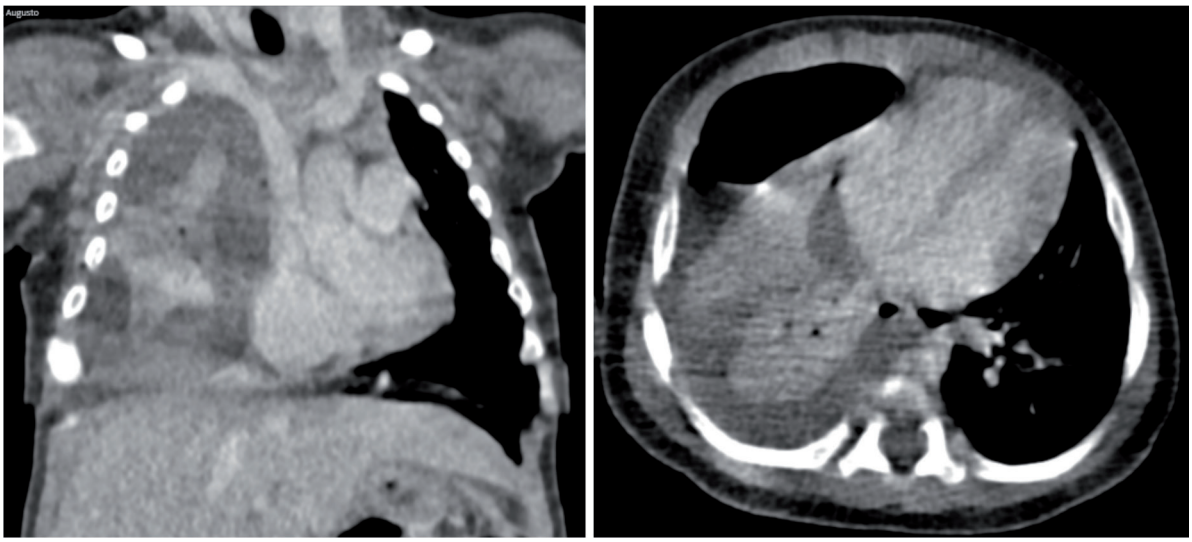

Figure 3. Day-2 chest CT demonstrated extensive consolidation involving nearly the entire right lung parenchyma, with areas of hypoattenuation and internal heterogeneity, consistent with necrotizing pneumonia. Multiple pneumatoceles were observed, the largest measuring 48 mm in the anterior region. A moderate right-sided pleural effusion, containing air bubbles, was also noted, producing compressive atelectasis of the adjacent lung.

40 mL of purulent fluid. MRSA was identified in pleural fluid samples, with molecular confirmation performed *via* PCR detection of the *mecA* gene and DNA amplification. PVL testing was negative. The admission blood culture was also negative. CSF cultures remained negative. Pleural fluid cultures confirmed MRSA susceptibility to vancomycin and clindamycin. Antimicrobial susceptibility testing was performed using automated methods and validated by disk diffusion, according to the European Committee on Antimicrobial Susceptibility Testing guidelines[4].

The patient showed progressive clinical and laboratory improvement, receiving supplemental oxygen for 48 hours (from day 2 to day 4), and becoming afebrile by day 5. The pleural drain was removed after 8 days, with a total output of 140 mL. No surgical debridement or intrapleural fibrinolytic therapy was required. Immunologic workup, including serum immunoglobulin levels, complement activity, and lymphocyte subsets, was unremarkable.

Clindamycin was administered for 7 days to provide toxin-suppressive activity, and vancomycin was continued for 28 days as definitive MRSA-directed therapy.

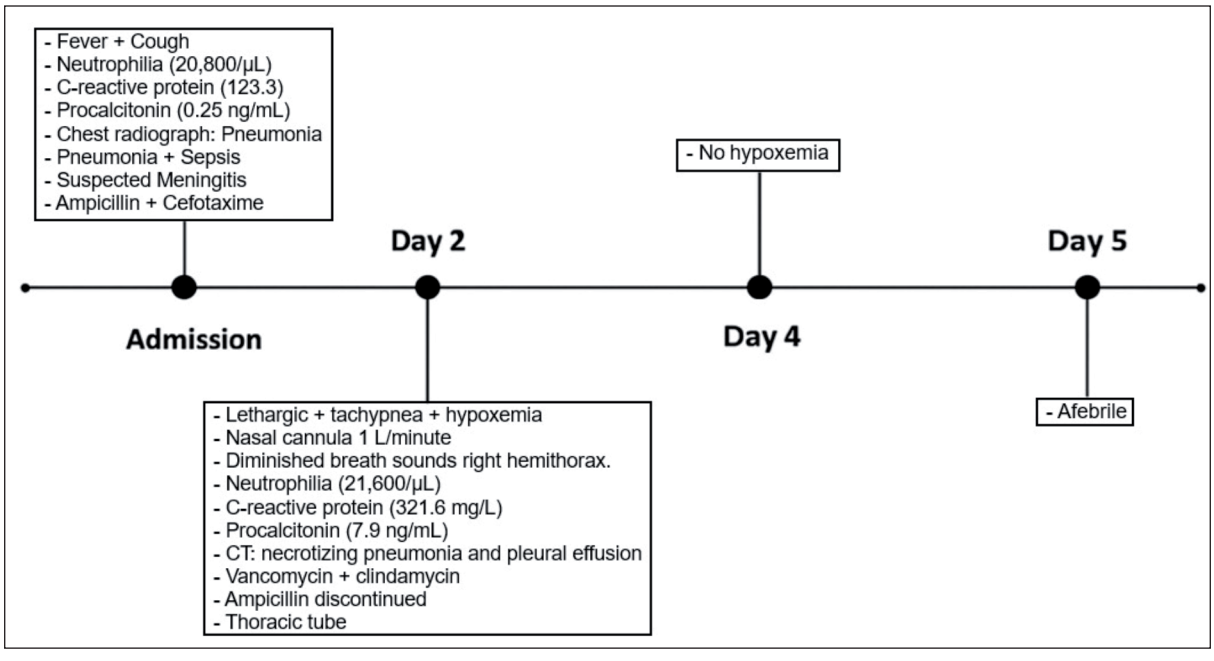

Figure 4. Timeline illustrating the patient's clinical course, laboratory findings, imaging results, and therapeutic interventions from admission until hypoxemia resolved and the patient became afebrile.

At discharge, a MRSA decolonization protocol was implemented. The patient completed a 5-day course of 2% mupirocin nasal ointment combined with topical cleansing using a solution containing 0.1% octenidine dihydrochloride and 2% 2-phenoxyethanol. The parents completed a 5-day MRSA decolonization regimen with 2% mupirocin nasal ointment and topical cleansing using 4% chlorhexidine gluconate solution.

DISCUSSION

CA-MRSA has emerged as an increasingly recognized pathogen in pediatric populations, capable of causing severe invasive diseases such as osteomyelitis and sepsis[5]. Although CA-MRSA pneumonia is uncommon in infants, reported cases underscore its potential for fulminant progression and significant morbidity[6,7].

In this case, the initial perineal bullous lesion likely served as the portal of entry. Infants have more vulnerable skin and mucosal barriers, facilitating hematogenous spread, even without underlying immunodeficiency[8,9]. Isolation of MRSA from the skin lesion and pleural fluid supports a direct temporal and microbiological link. This underscores the importance of early microbiological assessment of skin lesions in young infants.

When evaluating necrotizing pneumonia in this age group, it is important to acknowledge other potential etiologies such as *Streptococcus pneumoniae*, *Haemophilus influenzae*, and viral pathogens, including influenza and respiratory syncytial virus, which can predispose to secondary bacterial superinfection[10]. In this case, the clinical severity, the matching MRSA isolates from skin and pleural samples, and the absence of other pathogens on molecular testing supported MRSA as the causative agent.

Necrotizing pneumonia is characterized by parenchymal destruction and poor antibiotic penetration[11]. A high index of suspicion for MRSA is essential in cases of rapidly progressive pneumonia[12]. The patient's rapid deterioration led to early MRSA coverage, later confirmed by pleural fluid analysis. Rapid molecular diagnostic techniques are particularly valuable in these situations because cultures may be negative in up to 40% of cases due to prior antibiotic exposure. PCR testing provides fast and reliable MRSA identification in pleural samples, improving diagnostic accuracy even when conventional cultures fail[13]. Vancomycin remains the first-line treatment for invasive MRSA infections due to its reliable activity and ability to suppress toxin production[14,15].

Imaging findings such as pneumatoceles and air-fluid levels, observed in this case, are commonly associated with MRSA necrotizing pneumonia. While contrast-enhanced CT is often necessary to assess the extent of parenchymal destruction, guide drainage decisions, or identify complications such as bronchopleural fistulae, its routine use during treatment and recovery is not recommended. Chest radiographs should be used judiciously, with follow-up imaging guided by clinical course[16]. On the other hand, lung ultrasound may serve as a reliable, radiation-free diagnostic tool.

Management typically involves prolonged intravenous antibiotics and drainage of empyema when present. Unlike some reported cases[17,18] requiring intensive respiratory support or surgical intervention, this patient improved without the need for debridement therapy, highlighting the variability of disease severity despite the shared pathogen. Intrapleural fibrinolytic therapy may be considered for multiloculated effusions[19,20].

Finally, household decolonization is increasingly recommended to prevent recurrence and reduce transmission, particularly in cases involving CA-MRSA[21].

CONCLUSIONS

This case illustrates a severe presentation of necrotizing pneumonia in a 2-month-old infant caused by CA-MRSA. Even in the absence of underlying risk factors, primary skin infections may serve as a portal of entry for invasive disease. Early imaging with chest ultrasound and prompt microbiological diagnosis were critical in guiding appropriate management. In this patient, timely pleural drainage, without surgical debridement or fibrinolytics, was sufficient for recovery. Implementation of decolonization protocols for household contacts represents an important component of comprehensive management to reduce the risk of recurrence.

Conflict of Interest
The authors have no conflict of interest to declare.

Informed Consent
Written and informed consent was obtained from the parents for publication of this case report.

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None.

Authors' Contributions
Mário Ribeiro managed the patient, collected clinical data, and drafted the manuscript.
Marisa Coelho assisted in patient care and contributed to data collection and manuscript writing.
José Lopes provided specialized input and helped interpret findings.
Helena Silva and Augusta Gonçalves participated in patient follow-up and data acquisition.
Vera Baptista contributed to the literature review and critical revision of the manuscript.
Carmo Ferreira and Sofia Miranda supervised the case management and manuscript preparation, providing final approval of the version to be published.
All authors read and approved the final manuscript.

Ethics Approval
Not applicable.

Availability of Data and Materials
The data used to support the findings in this study are included within the article.

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AI Disclosure
No form of generative artificial intelligence was used for writing the manuscript.

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