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Invasive community-acquired MRSA infection causing osteomyelitis, endocarditis, and pulmonary embolism in an adolescent: a case report

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Case Report

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Abstract

Background

Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has emerged as an important cause of infection in otherwise healthy children and adolescents. Although most cases involve skin and soft-tissue infection, severe invasive disease—including osteomyelitis, endocarditis, and thromboembolic events—can occur and is associated with poor outcomes. In low- and middle-income countries, management is further challenged by nonspecific presentations and limited access to advanced diagnostic methods.

Case presentation:

A previously healthy 14-year-old adolescent presented with severe pain and swelling of the right lower limb following minor sports-related trauma, accompanied by fever. Evaluation revealed hematogenous osteomyelitis of the proximal femur complicated by deep vein thrombosis and pulmonary embolism. Blood cultures grew CA-MRSA and, based on the 2023 modified Duke criteria, infective endocarditis was diagnosed. The patient was treated with intravenous vancomycin and therapeutic anticoagulation, with a favorable clinical course.

Conclusions

This case underscores the challenges of recognizing and managing invasive CA-MRSA infections in resource-limited settings, where limited access to molecular testing and specialized imaging may delay diagnosis and targeted treatment. Early blood culture collection and prompt initiation of appropriate antimicrobial therapy are essential to reduce morbidity and mortality.

Background

Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) has emerged over recent decades as an important cause of infection in otherwise healthy individuals, including children and adolescents [1, 2]. Although most pediatric CA-MRSA infections involve skin and soft tissue, there has been a growing number of reports describing severe invasive disease, such as osteomyelitis, bacteremia, necrotizing pneumonia, endocarditis, and thromboembolic complications [3–5].

Population-based studies show a steady increase in pediatric CA-MRSA incidence, with estimated annual growth of up to 10% in some settings, reflecting a meaningful shift in the epidemiologic profile and increased vulnerability in this age group [6, 7]. Virulence factors such as Panton–Valentine leukocidin (PVL) have been associated with greater severity, including invasive and necrotizing forms of

disease [8]. In addition, some community strains exhibit resistance to non–β-lactam agents (e.g., clindamycin), further complicating therapeutic management [9].

Although international literature emphasizes early diagnosis and timely initiation of empirical antimicrobial therapy [7, 10], less is known about the availability of diagnostic methods and regional resistance patterns in low- and middle-income countries [11]. In Latin America, including Brazil, important gaps remain in knowledge regarding CA-MRSA epidemiology, particularly in pediatric populations [11, 12]. Rapid epidemiologic changes and the complexity of characterizing resistance phenotypes and virulence mechanisms in resource-limited contexts delay implementation of targeted treatment protocols based on local susceptibility patterns [12, 13].

We report a rare and severe case of invasive CA-MRSA infection in an adolescent complicated by thromboembolic events, highlighting diagnostic and therapeutic challenges in a public tertiary hospital in southern Brazil. We also review the regional literature to support local strategies for managing severe staphylococcal infections.

Case presentation

A previously healthy 14-year-old boy presented with severe pain and swelling of the right thigh and hip, with five days of fever. He reported minor trauma without impaired ambulation, described as a right lower-limb muscle strain after hyperextension during a soccer match. There had been no healthcare exposure or invasive procedures in the previous year. The patient reported recurrent furunculosis without complications and had not used antibiotics in the preceding three months.

On admission, vital signs were: axillary temperature 36.3°C, blood pressure 130/63 mmHg, heart rate 81 beats/min, and peripheral oxygen saturation 96% on room air. Physical examination revealed edema of the right thigh and calf and telangiectasias over the ipsilateral hip. No active or residual skin lesions were noted. There was no inguinal lymphadenopathy, and neurologic and vascular examinations were unremarkable. Bancroft, Homans, and Bandeira signs were negative. The left lower limb was normal. Cardiac auscultation disclosed a systolic murmur best heard at the mitral area, not previously documented.

Initial laboratory tests showed marked leukocytosis, an elevated C-reactive protein level (8.9 mg/dL), and an erythrocyte sedimentation rate of 70 mm/h. Given the initial suspicion of septic arthritis of the right hip, blood cultures were obtained and empirical intravenous ceftriaxone was initiated.

Venous Doppler ultrasound of the right lower limb demonstrated thrombosis of the common and superficial femoral veins. Chest computed tomography angiography was consistent with pulmonary embolism in segmental branches of the right lung (Fig. 1A and 1B). Magnetic resonance imaging showed hematogenous osteomyelitis of the proximal right femur, with no evidence of hip arthritis or soft-tissue collections (Fig. 2).

After four days of incubation, blood cultures grew oxacillin-resistant Staphylococcus aureus, and intravenous vancomycin was promptly initiated (1 g every 12 hours). Phenotypic characterization of the isolate was performed using the BD Phoenix™ M50 automated system based on the antimicrobial susceptibility profile and indirect resistance markers, with results interpreted according to BrCAST 2023 (version 13.0). Genotypic characterization was not available locally (e.g., detection of the *mecA* gene, PVL, and other virulence determinants). The isolate produced β-lactamase (BLACT positive) and showed a profile compatible with *mecA*-mediated resistance (MRS). It was susceptible to non–β-lactam agents, including daptomycin, erythromycin, gentamicin, linezolid, trimethoprim-sulfamethoxazole, and vancomycin, but resistant to clindamycin (Table 1).

Table 1
Antimicrobial susceptibility profile of Staphylococcus aureus isolated from blood culture.

Drug	MIC (mg/L)	Interpretation (BrCAST 2023)
Ciprofloxacin	≤ 0,5	I – Susceptible, increased exposure
Clindamycin	1	R – Resistant
Daptomycin	≤ 1	S – Susceptible
Erythromycin	≤ 0.25	S – Susceptible
Gentamicin	≤ 1	S – Susceptible
Levofloxacin	≤ 0.5	I – Susceptible, increased exposure
Linezolid	2	S – Susceptible
Oxacillin	> 2	R – Resistant
Penicillin G	> 1	R – Resistant
TMP-SMX	≤ 1/19	S – Susceptible
Vancomycin	1	S – Susceptible

Antimicrobial susceptibility testing was performed using the BD Phoenix™ M50 automated system, with interpretation according to BrCAST 2023 (version 13.0). Resistance markers detected: β-lactamase positive (BLACT) and *mecA*-mediated resistance (MRS). The category “I” (Susceptible, increased exposure) follows the EUCAST equivalence for adjusted breakpoints. TMP-SMX: trimethoprim-sulfamethoxazole.

Infective endocarditis was suspected because *S. aureus* bacteremia was accompanied by a cardiac murmur. Transthoracic echocardiography showed no vegetations, and transesophageal echocardiography was not available. The diagnosis was established using the 2023 modified Duke criteria, with one major criterion (positive blood cultures) and three minor criteria (fever, a new valvular murmur, and pulmonary embolism as a vascular phenomenon).

The patient received intravenous vancomycin for six weeks and therapeutic anticoagulation with enoxaparin (1 mg/kg every 12 hours). He was discharged after 35 days of hospitalization and continued anticoagulation with rivaroxaban. At outpatient follow-up, partial venous recanalization was documented and no infectious relapse occurred. Immunologic evaluation was normal, with no evidence of primary immunodeficiency or thrombophilia.

Discussion and Conclusions

We describe a previously healthy adolescent with recurrent furunculosis who developed severe invasive CA-MRSA infection, presenting with hematogenous osteomyelitis, infective endocarditis, deep vein thrombosis, and pulmonary embolism. The *S. aureus* isolate was resistance to β-lactam antibiotics, including oxacillin, and macrolides but remained susceptible to vancomycin, linezolid, and daptomycin. This pattern aligns with reports from Latin America, where more than 95% of CA-MRSA strains remain susceptible to glycopeptides and oxazolidinones, despite a gradual increase in resistance to quinolones and lincosamides [14].

Invasive CA-MRSA infections are uncommon but can be associated with high morbidity and mortality, particularly when multiple organ systems are involved [14, 16]. Pediatric studies suggest an association between invasive CA-MRSA infection and thromboembolic events, although the combination of hematogenous osteomyelitis and endocarditis is rare [15, 17]. *S. aureus* infection is an independent risk factor for venous thrombosis, with a reported risk up to 20-fold higher than in the general population [18]. Proposed mechanisms include systemic activation of coagulation, endothelial dysfunction, and increased expression of prothrombotic mediators; PVL may contribute to this process [19].

In this patient, local trauma may have facilitated bacterial entry in the setting of underlying cutaneous predisposition, leading to bacteremia followed by cardiovascular and thromboembolic complications. Early recognition of resistant staphylococcal infection and prompt initiation of targeted antimicrobial therapy are essential to prevent adverse outcomes [20]. However, in many low- and middle-income settings, limited access to rapid diagnostics and molecular testing delays recognition of these presentations. As a result, empirical therapy often relies heavily on clinical judgment, underscoring the importance of ongoing education to support timely recognition and appropriate management of CA-MRSA infections.

This case illustrates the practical challenges of rapidly diagnosing invasive CA-MRSA infections in resource-limited settings, where nonspecific symptoms can mimic benign musculoskeletal conditions, particularly in otherwise healthy patients with minor trauma. The absence of overt skin lesions and restricted access to advanced tests—such as early magnetic resonance imaging, transesophageal echocardiography, and rapid microbiological assays—may delay recognition of bacteremia and its thromboembolic complications.

In contrast to high-income countries where genomic surveillance is widespread, Latin America has substantial gaps in CA-MRSA surveillance [21]. In Brazil, few studies with representative samples have

assessed MRSA prevalence in community-acquired infections, and the lack of routine molecular testing for the *mecA* and PVL genes likely leads to underestimation of the true burden. In southern Brazil, studies have reported high oxacillin resistance rates, averaging above 40% among community isolates [11]. Reported CA-MRSA prevalence varies widely—generally 10% to 40% of community *S. aureus* isolates—with clones related to international lineages and increasing overlap between community and hospital profiles [22]. This heterogeneity reflects both true epidemiologic variation and limitations in microbiological surveillance, reinforcing the need for multicenter studies to better estimate the prevalence of CA-MRSA infection in Brazil.

Close collaboration with the infectious diseases team enabled timely, appropriate care. Initiation of intravenous vancomycin immediately after MRSA identification is consistent with international guidance for MRSA bacteremia and osteomyelitis, which generally recommends 4–6 weeks of intravenous therapy for invasive infection [23]. Similarly, therapeutic-dose enoxaparin followed by an oral anticoagulant is consistent with available evidence for the management of infection-associated venous thromboembolism in children [18, 19].

This report highlights the potential severity of CA-MRSA infections in children and adolescents, which may extend beyond typical cutaneous disease to invasive, life-threatening infection with thromboembolic complications. In settings with diagnostic limitations, strong clinical suspicion, early drawing of blood cultures, and prompt initiation of appropriate antimicrobial therapy are critical determinants of prognosis. Strengthening microbiological surveillance, expanding access to molecular methods, and generating regional pediatric-focused epidemiologic data are essential to optimize clinical management and reduce CA-MRSA-associated morbidity and mortality in low- and middle-income countries.

Abbreviations

CA-MRSA: community-acquired methicillin-resistant *Staphylococcus aureus*; MRSA: methicillin-resistant *Staphylococcus aureus*; PVL: Panton–Valentine leukocidin; DVT: deep vein thrombosis; PE: pulmonary embolism; MIC: minimum inhibitory concentration; TMP-SMX: trimethoprim-sulfamethoxazole.

Declarations

Ethics approval and consent to participate: Not applicable.

Consent for publication: Written informed consent for publication of this case report and any accompanying images was obtained from the patient's parent.

Availability of data and materials: All available information is contained within the manuscript.

Competing interests: The authors declare no competing interests.

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Authors' contributions: CH conceived the case report and supervised clinical management. ACCM and MCM collected and organized the clinical data. LCTF analyzed and interpreted the patient's data related to the imaging exams. BHS and RKNC contributed to the interpretation of microbiological/pharmacological data and critically reviewed the manuscript. CH, ACCM, and MCM performed the literature review. ACCM and MCM drafted the first version of the manuscript. All authors critically revised the manuscript and approved the final version.

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Figures

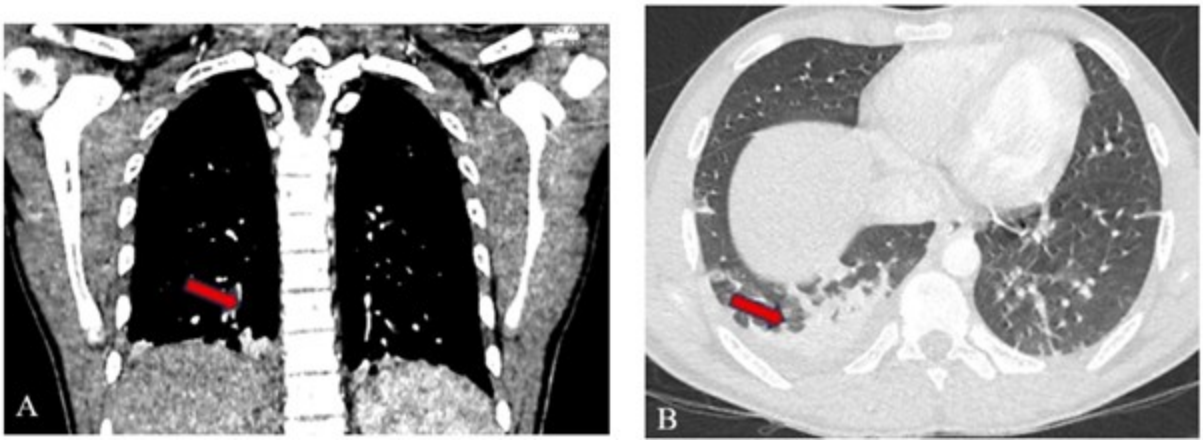


Figure 1

A: Filling defect in the posterior basal segmental branch (arrow) of the right lung; B: Underlying heterogeneous subpleural pulmonary consolidation (arrow).

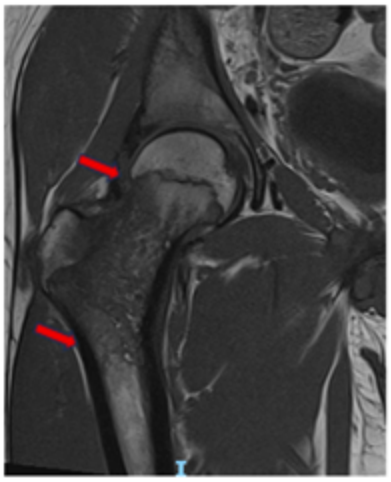


Figure 2

Magnetic resonance imaging showing bone marrow edema (arrows) in the proximal femur, consistent with acute osteomyelitis.