CASE REPORT

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From a minor skin lesion to an invasive *Staphylococcus aureus* infection in an adolescent

De una lesión leve de piel a una infección invasiva por Staphylococcus aureus *en un adolescente*

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ABSTRACT

Introduction: *Staphylococcus aureus* is a common causative agent of skin and soft tissue infections in pediatric populations. However, it can also lead to invasive diseases such as osteoarticular infections, pneumonia, and bacteremia. This case report describes the progression of a superficial skin infection in an adolescent that evolved into a severe invasive condition. **Case presentation:** a 16-year-old previously healthy patient presented with a skin lesion in the cervical region. Within days, the infection rapidly progressed, leading to fever, malaise, and systemic symptoms. Blood cultures confirmed methicillin-resistant *S. aureus* (MRSA), and imaging studies revealed pulmonary septic emboli. The patient was started on broad-spectrum antibiotics, adjusted according to susceptibility testing. Clinical improvement was observed, and the patient was discharged without complications. **Conclusion:** this case highlights the potential for *S. aureus* to progress rapidly from a minor skin infection to a life-threatening invasive disease in pediatric patients. It underscores the importance of early recognition and appropriate management to prevent severe complications.

Keywords: *Staphylococcus aureus*, pediatric infections, bacterial resistance, methicillin-resistant *Staphylococcus aureus*.

RESUMEN

Introducción: Staphylococcus aureus *es un agente causal común de infecciones de piel y tejidos blandos en la población pediátrica. Sin embargo, puede provocar enfermedades invasivas como infecciones osteoarticulares, neumonía y bacteriemia. Este caso clínico describe la progresión de una infección cutánea superficial en un adolescente que evolucionó a una afección invasiva grave.* ***Presentación del caso:*** *paciente de 16 años, previamente sano, quien presentó una lesión cutánea en la región cervical. En cuestión de días, la infección progresó rápidamente, provocando fiebre, malestar general y síntomas sistémicos. Los hemocultivos confirmaron la presencia de S. aureus resistente a la meticilina, y los estudios de imagen revelaron émbolos pulmonares. Se inició tratamiento con antibióticos de amplio espectro, ajustados según las pruebas de sensibilidad. Se observó mejoría clínica y el paciente fue dado de alta sin complicaciones.* ***Conclusión:*** *este caso destaca la posibilidad de que en pacientes pediátricos con infección cutánea por S. aureus progresen a una enfermedad invasiva potencialmente mortal. Por lo que es importante su reconocimiento y el tratamiento apropiados para prevenir complicaciones graves.*

Palabras clave: Staphylococcus aureus, *infecciones en pediatría, resistencia bacteriana,* Staphylococcus aureus *meticilino resistente.*

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Abbreviations:
CA-MRSA = community-acquired methicillin-resistant *S. aureus*.
CRP = C-reactive protein.
ESR = erythrocyte sedimentation rate.
MIC = Minimum inhibitory concentrations.
MRSA = methicillin-resistant *Staphylococcus aureus*.
PT = prothrombin time.
PTT = partial thromboplastin time.
PVL = Panton-Valentine leukocidin.
SSTI = skin and soft tissue infections.

INTRODUCTION

Staphylococcus aureus (S. aureus) is one of the most common causes of skin and soft tissue infections in pediatric populations. If not treated appropriately, these infections can progress to more severe conditions. This pathogen can cause a broad spectrum of illnesses, ranging from skin abscesses to potentially life-threatening diseases such as bacteremia, pneumonia, and endocarditis.[1]

The progression to severe forms is influenced not only by individual patient factors –such as immunosuppressive conditions or genetic predispositions– but also by the emergence of antibiotic-resistant strains, including methicillin-resistant *Staphylococcus aureus* (MRSA) and community-acquired MRSA (CA-MRSA).[1,2]

Several studies have documented CA-MRSA infections in children, underscoring its high virulence and rapid capacity for dissemination. For instance, a retrospective study by Padilla et al.[3] described a series of cases in which initial skin infections quickly progressed to bacteremia and systemic complications. Similarly, Morris et al.[4] reported the rapid evolution of CA-MRSA skin infections into septic embolism in a cohort of previously healthy children. Moreover, recent research has highlighted the role of specific host genetic factors in increasing susceptibility to severe systemic infections.[5]

To underscore the importance of timely management of MRSA infections in pediatric patients, this case report presents an adolescent who developed a severe MRSA infection that progressed from a skin lesion to bacteremia and pulmonary septic embolism.

CASE PRESENTATION

A 16-year-old male with no known medical, surgical, or allergic history, and with adequate nutritional habits, presented with a 15-day history of symptoms. His family history was notable for diabetes mellitus in both grandparents.

The clinical course began with the appearance of a pruritic papule –an elevated, erythematous, and itchy lesion– on the posterior cervical region. The lesion progressively worsened, exhibiting increased erythema, warmth, tenderness, and eventually leading to spontaneous purulent drainage. These local symptoms were accompanied by persistent fever, ranging from 39 to 40 °C. At a previous hospital, the patient was treated with doxycycline for four days without clinical improvement. He subsequently sought care at another institution, where he was prescribed a seven-day course of outpatient clindamycin. However, he returned due to persistent fever and the onset of torticollis.

Upon admission to our hospital, the patient's vital signs were as follows: blood pressure 109/60 mmHg, heart rate 128 beats per minute, respiratory rate 19 breaths per minute, oxygen saturation 88%, and a pain score of 1/10. Anthropometric measurements included a weight of 49.8 kg, height of 155 cm, and a body mass index (BMI) of 20.73 kg/m². Physical examination revealed no signs of malnutrition and a generally stable condition. An infected wound was noted on the posterior neck, characterized by warmth, erythema, tenderness, edema, and yellow purulent discharge. The lesion exhibited features consistent with an abscess and fibrinoid tissue, without evidence of muscle or bone exposure.

Laboratory tests revealed leukocytosis, with a white blood cell count of 14,900 cells/mm³, predominantly neutrophilic (12,700 cells/mm³). Lymphocyte and monocyte counts were 1,400 cells/mm³ and 700 cells/mm³, respectively. Hemoglobin was 16.3 g/dL, and the platelet count was 177,000 cells/mm³. Coagulation times were within normal limits. C-reactive protein was elevated at 45.1 mg/dL, while the erythrocyte sedimentation rate was 16 mm/h. Renal function tests showed a creatinine level of 0.8 mg/dL and a blood urea nitrogen of 13 mg/dL, with an estimated glomerular filtration rate of 106 mL/min/1.73 m². Serologic testing for human immunodeficiency virus (HIV) was non-reactive.

The abscess culture yielded growth of MRSA. Antimicrobial susceptibility testing revealed resistance to clindamycin (MIC > 4 µg/mL) and oxacillin (MIC > 2 µg/mL), while the isolate remained susceptible to linezolid (MIC = 2 µg/mL), trimethoprim/sulfamethoxazole (MIC ≤ 0.5/9.5 µg/mL), and vancomycin (MIC = 1 µg/mL). Two blood cultures were also positive for *Staphylococcus*

aureus, with susceptibility profiles consistent with those of the abscess isolate. Susceptibility testing was performed using the MicroScan system (Baxter) and interpreted with WHONET 5.6 software (World Health Organization). Minimum inhibitory concentrations (MICs) were interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines.

Treatment included isotonic crystalloids, oxygen therapy, acetaminophen, and intravenous vancomycin (500 mg every six hours), with therapeutic drug monitoring maintained within the recommended range of 5-10 µg/mL. On the third day of hospitalization, the patient developed signs of systemic inflammatory response, hypoxemia, and respiratory distress. A chest X-ray *(Figure 1)* revealed left lung consolidation and pleural effusion with an interpleural distance of 13 mm, without evidence of septations. Open thoracotomy or decortication was not deemed necessary. Four days later, a follow-up pleural ultrasound showed resolution of the pleural effusion. A contrast-enhanced neck CT scan *(Figure 2)* demonstrated inflammatory changes in the soft tissues on the left side of the neck, with muscle thickening and findings consistent with pulmonary septic embolism.

The patient showed favorable clinical progression and completed a 14-day course of vancomycin. He never required inotropic support, mechanical ventilation, or admission to the pediatric intensive care unit. Despite persistent *S. aureus* bacteremia in three sets of blood cultures during the first six days, subsequent cultures turned negative. A single transthoracic echocardiogram performed during hospitalization was normal, with no evidence of vegetations. Regarding the abscess, the surgical team determined that drainage was not necessary. Over the course of two weeks, it was successfully managed only with topical treatment.

DISCUSSION

Skin and soft tissue infections caused by *S. aureus* represent the most common clinical manifestation associated with this pathogen. CA-MRSA accounts for approximately 30 to 40% of these cases. This trend is particularly relevant in the pediatric population, where CA-MRSA infections have shown increasing frequency and severity over recent decades. *S. aureus* has developed a high level of resistance to methicillin and other beta-lactam antibiotics, making it a significant cause of both community, and healthcare-associated infections.[1-3]

The occurrence and severity of *S. aureus* infections are influenced by a combination of host, pathogen, and environmental factors. Host-related factors include the child's genetic background, age, sex, immune status, nutritional condition, and the presence of underlying health issues. These elements significantly affect the body's ability to mount an effective immune response. Pathogen-related factors involve the virulence of the infectious agent, the intensity and mode of transmission, its adaptability to the host environment, and its resistance to antimicrobial agents. Virulence factors, in particular, facilitate colonization, tissue invasion, and cellular damage. Environmental factors such as sanitation, air and water quality, ambient temperature, housing conditions, hygiene practices, and exposure to colonized individuals (e.g., in daycare

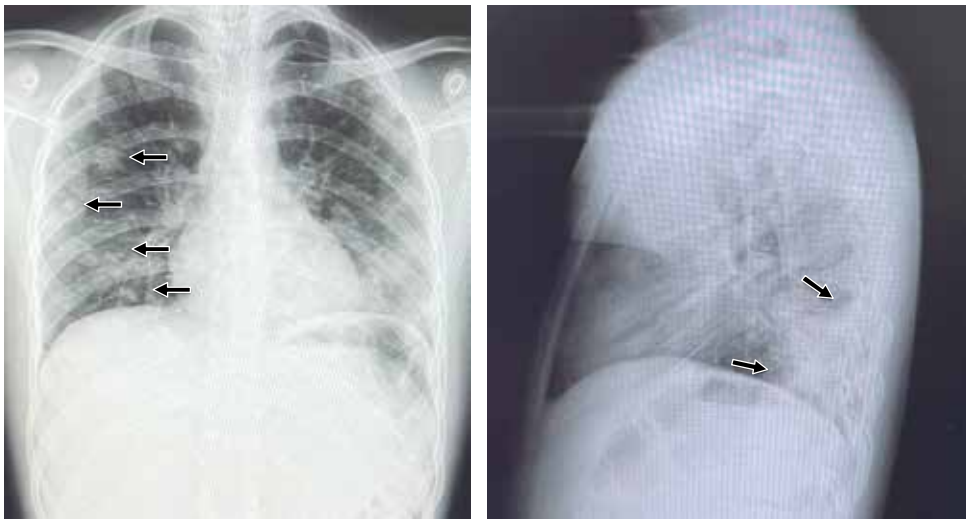

Figure 1:

Chest X-ray, anteroposterior (AP) and lateral projections. Multiple parenchymal opacities with pseudonodular morphology are observed, randomly distributed and predominantly located in the peripheral regions of the middle third of the right lung field. These findings are associated with left basal consolidation. A small left pleural effusion is also noted (black arrows).

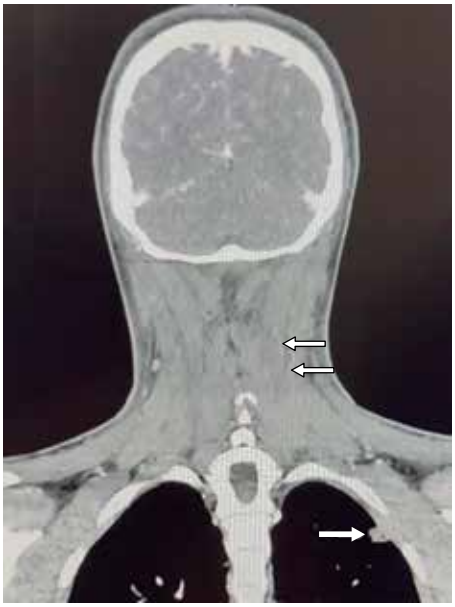

Figure 2: Contrast-enhanced neck CT scan. Evidence of soft tissue discontinuity in the left posterolateral region of the neck, with a hypodense area, thickening of the muscular planes, and altered density of the surrounding fat tissue. Cutaneous thickening is also noted. Additionally, a nodular lesion is visible in the upper thoracic region, suggestive of a septic pulmonary embolism (white arrows).

settings) also play a critical role in infection risk and progression.[2-7]

Shilo et al. have reported a significant increase in the incidence of complicated infections in pediatric populations, coinciding with the rise of CA-MRSA infections. CA-MRSA primarily affects previously healthy children –as observed in our case– suggesting a potentially more aggressive clinical behavior. For example, CA-MRSA pneumonia is often associated with extensive lung necrosis, a phenomenon attributed to the production of pore-forming toxins, particularly Panton-Valentine leukocidin (PVL) and alpha-hemolysin.[8]

In the present clinical case, it is noteworthy that the patient –previously healthy– presented with a soft tissue infection that progressed to persistent bacteremia and pulmonary septic embolism, raising suspicion of a highly virulent *S. aureus* strain. This pathogen employs a broad arsenal of virulence factors, including toxins and resistance genes, which contribute to its pathogenic potential. Among others, key virulence mechanisms include adhesion, bacterial invasion, protease secretion, immune evasion and extracellular matrix degradation. These factors collectively enhance the bacterium's ability to disseminate through the bloodstream and invade distant tissues, as observed in this case.[9-11]

In recent years, MRSA has emerged in community settings, causing infections in previously healthy individuals without identifiable exposure to healthcare environments. CA-MRSA clones, such as USA300, are genetically distinct from hospital-associated strains. The USA300 clone, derived from USA500, harbors a bacteriophage encoding PVL, a pore-forming cytotoxin that targets and lyses human neutrophils. This clone is frequently isolated in CA-MRSA infections and is strongly associated with deep soft tissue infections –including boils, furunculosis, and abscesses– as well as life-threatening conditions such as necrotizing pneumonia, bacteremia, osteomyelitis, and endocarditis. Its enhanced virulence and transmissibility have made USA300 a dominant lineage in many regions.[12-14]

An observational and prospective study conducted between 2006 and 2007 across seven hospitals in three Colombian cities confirmed the circulation of three predominant MRSA clones in the country. These included: (1) a Chilean clone, (2) a pediatric clone associated with hospital-acquired MRSA infections, and (3) a community-associated clone related to USA300, carrying the SCCmec IVc element. The identification of the USA300-related clone in community settings underscores the regional spread of highly virulent CA-MRSA strains and their capacity to cause severe infections in previously healthy individuals. This finding emphasizes the importance of considering bacterial virulence genes in similar clinical scenarios.[15-20]

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