American Journal of Emergency Medicine (2010) **28**, 537.e3–537.e6

The American Journal of Emergency Medicine

www.elsevier.com/locate/ajem

Case Report

A rare case of pyomyositis complicated by compartment syndrome caused by ST30–staphylococcal cassette chromosome *mec* type IV methicillin-resistant *Staphylococcus aureus*☆

Abstract

Diseases caused by community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) are frequently associated with skin and soft tissue infections. However, unusual life-threatening infections in immunocompetent children and young adults have increasingly been reported, such as necrotizing fasciitis, pyomyositis, septic thrombophlebitis of the extremities, rapidly progressive and necrotizing pneumonia, acute hematogeneous osteomyelitis, septic thrombophlebitis, and Waterhouse-Friderichsen syndrome. In the case reported here, we describe a rare case of traumatic pyomyositis complicated with compartment syndrome in an immunocompetent 8-year-old child. Using staphylococcal cassette chromosome *mec* typing, pulsed-field gel electrophoresis and multilocus sequence typing, it was found that the CA-MRSA isolate recovered belonged to ST30–staphylococcal cassette chromosome *mec* type IV lineage. In addition, polymerase chain reaction amplifications showed that this isolate was a Panton-Valentine leucocidin-producer CA-MRSA and also harbors the enterotoxin gene cluster locus (*egc*). Therefore, it is important that physicians be aware that CA-MRSA must be considered in case of deep skeletal muscle infections complicated with compartment syndrome.

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a leading cause of hospital infection. After the late 1990s, increasing cases of community-associated MRSA (CA-MRSA) diseases have been reported in different countries [1]. Many of CA-MRSA isolates display staphylococcal cassette chromosome *mec* (SCC*mec*) type IV or V, Panton-Valentine leucocidin (PVL), and ample susceptibility to non–β-lactam antimicrobials [2]. In Brazil, the lineage ST30-SCC*mec*IV (Oceania Southwest Pacific clone) has been associated to CA-MRSA diseases in Porto Alegre, RS, and in Rio de Janeiro, RJ [3,4]. Although most CA-MRSA diseases are restricted to skin/soft tissue infections, some cases can be complicated by severe manifestations including necrotizing pneumonia, endocarditis, necrotizing fasciitis, and generalized osteomyelitis. In the case reported herein, we describe a rare case of forearm pyomyositis associated to traumatic injure in an immunocompetent 8-year-old boy complicated by compartment syndrome and severe pneumonia caused by a CA-MRSA isolate of the ST30-SCC*mec*IV lineage.

A healthy 7-year-old boy, living in Rio de Janeiro City, with a history of traumatic injury, had an initial diagnostic of muscle contusion of the left forearm and had been immobilized 3 days after the trauma. The boy presented to the emergency department of our hospital 2 days after the immobilization with a history of progressive pain in the left forearm, high fever, and generalized myalgia. The patient was septic, prostrated, dehydrated, and with high temperature (>39°C). He presented with tachycardia (150 beats/min), hyperventilation of 80 breaths/min, and hepatomegaly. The physical examination of the affected area revealed significant edema, warmth, induration in the left elbow area, and intense pain. Blood samples were collected for hemocultures, and the patient was given cefepime and clindamycin.

In the next day, after the diagnostic of compartment syndrome, the patient was submitted to fasciotomy of left forearm and elbow arthrotomy. The drained purulent secretion was also collected for bacterial cultures. Immediately after surgery, the child was sent to the pediatric intensive care unit in severe conditions with oxygen therapy (mask reservoir at a driving gas flow of 8 L/min), presenting with refractory hypotension and signs and symptoms of systemic inflammatory response syndrome. The thoracic radiograph showed diffuse infiltrate in both lungs, predominantly in the right (Fig. 1A). The patient was treated with amine-vasopressors (dobutamine and noradrenaline) and hydrocortisone. Few hours later, the boy progressed to a state of disorientation, metabolic acidosis (bicarbonate 15.8 mmol/L and pH 7.18), tachypnea, high fever (39°C), and purple papules on the members and body. The patient developed acute lung injury with evolution to acute

☆ This work was supported in part by Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (FAPERJ) e Fundação Conselho de Aperfeiçoamento de Pessoal do Ensino Superior (CAPES). FAF and MSR were recipients of fellowships granted by CNPq.

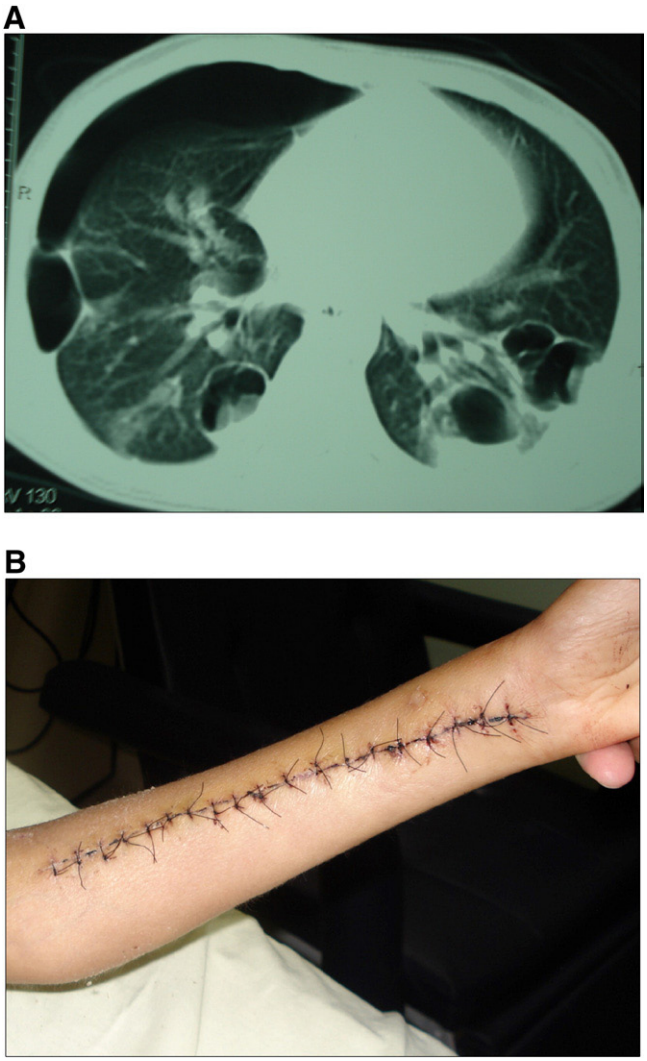

Fig. 1 A, Multiple bullous lesions suggestive of *S aureus* infection were evident in lung computed tomography. B, Postsurgical picture of the child's left forearm demonstrating the extension of the lesion.

respiratory distress syndrome in 5 hours. Orotracheal intubation and mechanical ventilation were required.

Two days after intensive care unit admission, the patient was more conscious but still hemodynamically unstable and tachycardic. He developed anemia (Ht, 25%, and Hb, 9.0 g/dL) and had a leukocytosis count of 35 000/μL, 14% band neutrophils, blood platelets of 80 000/μL, and prothrombin time value of 66%, with poor peripheral perfusion, generalized edema, and a yellowish tracheobronchial secretion. Echocardiogram was normal, and chest radiographs showed right-sided hypertensive pneumothorax that was treated with a chest drain insertion. Blood was again collected for hemocultures, and the empiric therapy was switched to vancomycin, meropenem, and amikacin.

In the next day, the child was evaluated by a vascular surgeon because of the increase of the forearm edema, and examination showed cold left extremities, poor perfusion, and absence of ulnar and radial pulses. The Doppler fluxometry of the left forearm revealed triphasic flux for the ulnar and absence of flux in the radial arteries, indicating the need of another surgery. The patient underwent fasciotomy, with discharge of a huge amount of purulent secretion from the anterior portion of the left forearm and necrosis of the deep flexor muscle of fingers. The lesion was drained, and debridement of the necrotized tissue performed.

Next day after surgery, the patient condition progressively improved, and the child showed better temperature (37.8°C) and more stable blood pressure; the edema diminished with improvement of the tissue perfusion, and the patient was responding to the stimulus. The new echocardiogram was also normal. The C-reactive protein was 4.7 mg/L; leukocyte counts, 20 600/μL; 6% band neutrophils; and Ht, 27.5%. The better respiratory parameters allowed the reduction of vasopressors and hydrocortisone. Because the *S aureus* isolates recovered were resistant to methicillin, the antimicrobial therapy with amikacin and meropenem was discontinued and vancomycin maintained. After 8 days in the intensive care unit, the hemocultures were negative. On day 10, lesion in the left forearm was sutured (Fig. 1B) and the extubation proceeded, with maintenance of the O_2 mask and thoracic drain, which was removed 2 days after.

After 20 days of vancomycin therapy, the fever reoccurred, and the laboratorial examinations showed leukocytes counts of 20 600 μL; 7% band neutrophils; and C-reactive protein, 9.0 mg/L. The thoracic radiograph images showed air-fluid level at the lung apices and base. The computed tomography of the left forearm showed bone refraction in the one third portion of radio, and bone scintilography showed hypercaptation in the radio area, with inconclusive diagnostic for osteomyelitis, due to the previous surgical manipulations in the area. After these considerations, the therapy was switched to linezolid. The patient presented progressive improvement of the clinical conditions, the fever has effectively disappeared, the pulmonary disease has improved, the left forearm has cicatrized, and C-reactive protein was 1.1 mg/L. The linezolid was discontinued after a 31-day treatment. In the 57th day of hospitalization, the patient who only showed a slight difficulty of extending the left hand was discharged to attend ambulatorial follow-up visits, with recommendation of physiotherapy treatment.

The *S aureus* isolates were identified by routine methods. Antimicrobial susceptibility testing was carried out as recommended by the Clinical Laboratories Standard Institute [5] using the following antimicrobial disk: 1 μg oxacillin, 5 μg ciprofloxacin, 30 μg chloramphenicol, 2 μg clindamycin, 15 μg erythromycin, 10 μg gentamicin, 5 μg rifampin, 125 μg/23.75 μg sulfametoxazol-trimetroprim, and 30 μg vancomycin. The *mecA* gene and the gene *lukF*-pv (encoding the LukF subunit of the leukocidin) were assessed by polymerase chain reaction (PCR), as described [3]. SCC*mec* typing was carried out by multiplex PCR [6]. The bacterial genomic DNA was cut with *Sma*I, and the fragments were

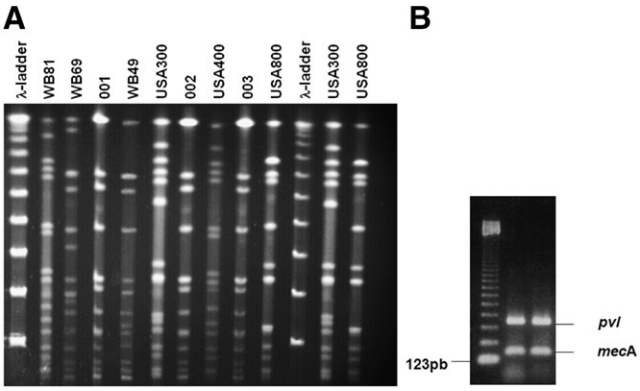

Fig. 2 A, Pulsed-field gel electrophoresis of *Sma*I-fragmented genomic DNA. Lines 1 and 11: λ-Ladder molecular size marker; lines 2 and 7: WB81 and USA400 are CA-MRSA representatives of the lineage ST1-SCC*mec*IV; lines 3 and 5: WB69 and WB49 are CA-MRSA representatives of the lineage ST30-SCC*mec*IV; lines 4, 7, and 9: 001, 002, and 003 are CA-MRSA isolates recovered from blood cultures of the case reported (note that the PFGE pattern is quite identical to the isolate WB49); lines 6 and 12: a representative of the USA300 CA-MRSA (ST9-SCC*mec*IV) widespread in the United States; lines 10 and 13: USA800 is a representative of the hospital-associated lineage ST5-SCC*mec*IV, frequently found in this country. B, Polymerase chain reaction amplifications of internal fragments of *lukF*-pv (codifying for the F-subunit of PVL) and *mecA* gene (determining methicillin resistance).

separated by pulsed-field gel electrophoresis (PFGE) [7]. The multilocus sequencing typing was performed as assigned by www.mlst.net. Polymerase chain reaction for amplifying internal fragments of *seg* and *sen* genes was carried out as previously described [3] to assess the presence of enterotoxin gene cluster (*egc*). Also, the *tst* (encoding for toxic shock syndrome toxin 1) was also assessed by PCR [3].

It was found that the MRSA isolates carried SCC*mec* type IV and were susceptible to all other non–β-lactam antimicrobial tested. In addition, the isolates showed a PFGE pattern very similar (1 band difference) to that of the CA-MRSA representative of the lineage ST30-SCC*mec* IV, previously detected in our country causing skin/soft tissue infections and septic arthritis [3,4] (Fig. 2A) (Fig. 1A). The MRSA isolates were typed as ST30 by multilocus sequencing typing. The presence of *lukF*-pv gene (Fig. 2B) and of *egc* locus, common in this CA-MRSA lineage, were confirmed, but *tst* was not detected. The presence of *egc* locus, common in this CA-MRSA lineage, was confirmed, but *tst* was not detected.

Pyomyositis is an infection of skeletal muscle mostly due to *S aureus*. Tropical pyomyositis is rare and characterized by the presence of abscess [6]. Despite the fact bacterial myositis is more seen in humid areas, nontropical pyomyositis is of increasing recognition in temperate climates [8]. The diagnosis of pyomyositis is often overlooked because most physicians are not familiar with this entity. Indeed, the pathogenesis of bacterial myositis is not well understood [9]. Local damage seems to be an important factor in the condition of transient bacteremia. However, nontropical pyomyositis seems to be more associated with immunocompromised patients [9]. The increased incidence of pyomyositis seems to have paralleled by the emergence of CA-MRSA. In the United States, CA-MRSA was the most frequent pathogen accounting for 35% of the cases [10]. However, compartment syndrome as a complication of staphylococcal pyomyositis is very rare. The first case of pyomyositis convoluted by compartment syndrome was described in 2008, associated with USA300 (ST8-SCC*mec*IV) isolate that affected a young woman who, as the boy of the case reported here, did not present risk factors for MRSA infections [11]. To our knowledge, the case reported here is the second reported case of pyomyositis complicated by compartment syndrome, associated to CA-MRSA.

Infections caused by CA-MRSA are escalating in incidence worldwide [12]. Although there is an intensive discussion as to whether CA-MRSA is more virulent that methicillin susceptible *S aureus*, there is no conclusive study on such topic. In addition, the role of PVL in the virulence of CA-MRSA infections is still subject of debate [13]. Despite that, there are some evidences that CA-MRSA infections may have a particularly rapid progress [13,14]. Thus, it is imperative that physicians should be aware of the circulation of PVL-producer CA-MRSA strains causing both rare and complicated infections, so that the anti–CA-MRSA therapy could be promptly installed in cases of extremely severe staphylococci infections.

Bruno Espirito Santo de Araújo MD
Centro de Terapia Intensiva Pediátrica e Neonatal
Hospital Central da Polícia Militar do Rio de Janeiro
Rio de Janeiro, Brazil

Juliana Milward Borchert MD
Centro de Controle de Infecção Hospitalar
Hospital Central da Polícia Militar do Rio de Janeiro
Rio de Janeiro, Brazil

Paulo Gustavo Manhães MD
Serviço de Ortopedia
Hospital Central da Polícia Militar do Rio de Janeiro
Rio de Janeiro, Brazil

Fabienne Antunes Ferreira
Mariana Severo Ramundo
Maria Cíceras Silva-Carvalho
Universidade Federal do Rio de Janeiro
Instituto de Microbiologia Paulo de Góes
Rio de Janeiro, Brazil

Antonio Carlos Seabra MD
Sandra Helena Victal MD
Centro de Terapia Intensiva Pediátrica e Neonatal
Hospital Central da Polícia Militar do Rio de Janeiro
Rio de Janeiro, Brazil

Agnes Marie Sá Figueiredo PharmD, PhD
Universidade Federal do Rio de Janeiro
Instituto de Microbiologia Paulo de Góes
Rio de Janeiro, RJ, Brazil
E-mail address: agnes@micro.ufrj.br

doi:10.1016/j.ajem.2009.08.010

References

[1] Diep BA, Otto M. The role of virulence determinants in community-associated MRSA pathogenesis. Trends Microbiol 2008;16:361-9.
[2] Nygaard TK, DeLeo FR, Voyich JM. Community-associated methicillin-resistant *Staphylococcus aureus* skin infections: advances toward identifying the key virulence factors. Curr Opin Infect Dis 2008;21:147-52.
[3] Ribeiro A, Dias C, Silva-Carvalho MC, Berquó L, Ferreira FA, Santos RN, et al. First report of infection with community-acquired methicillin-resistant *Staphylococcus aureus* in South America. J Clin Microbiol 2005;43:1985-8.
[4] Ribeiro A, Coronado AZ, Silva-Carvalho MC, Ferreira-Carvalho BT, Dias C, Rozenbaum R, et al. Detection and characterization of international community-acquired infections by methicillin-resistant *Staphylococcus aureus* clones in Rio de Janeiro and Porto Alegre cities causing both community- and hospital-associated diseases. Diagn Microbiol Infect Dis 2007;59:339-45.
[5] CLSI - Clinical and laboratory standards institute. Performance standards for antimicrobial disk susceptibility tests. 9th edn. Approved Standard M2-A9. Wayne: CLSI document M100-S17; 2007.
[6] Oliveira DC, de Lencastre H. Multiplex PCR strategy for rapid identification of structural types and variants of the mec element in methicillin-resistant *Staphylococcus aureus*. Antimicrob Agents Chemother 2002;46:2155-61.
[7] Soares MJ, Teixeira LA, Nunes MR, da Silva Carvalho MC, Ferreira-Carvalho BT, Figueiredo AM. Analysis of different molecular methods for typing methicillin-resistant *Staphylococcus aureus* isolates belonging to the Brazilian epidemic clone. J Med Microbiol 2001;50:732-42.
[8] Torres M, Rosati JLR, Gripp AC. Piomiosite tropical. In: Talhari S, Garrido Neves R, editors. Dermatologia tropical. Rio de Janeiro, RJ: Ed Medsi 1997, p. 305-7.
[9] Lupi O, Madkan V, Tyring SK. Tropical dermatology: bacterial tropical diseases. Am Acad Dermatol 2006;54:559-78.
[10] Pannaraj PS, Hulten KG, Gonzalez BE, Mason Jr EO, Kaplan SL. Infective pyomyositis and myositis in children in the era of community-acquired, methicillin-resistant *Staphylococcus aureus* infection. Clin Infect Dis 2006;43:953-60.
[11] Shedek BK, Nilles EJ. Community-associated methicillin-resistant *Staphylococcus aureus* pyomyositis complicated by compartment syndrome in an immunocompetent young woman. Am J Emerg Med 2008;26:737.e3-e4.
[12] Moellering Jr RC. Current treatment options for community-acquired methicillin-resistant *Staphylococcus aureus* infection. Clin Infect Dis 2008;46:1032-7.
[13] Diep BA, Palazzolo-Ballance AM, Tattevin P, Basuino L, Braughton KR, Whitney AR, et al. Contribution of Panton-Valentine leucocidin in community-associated methicillin-resistant *Staphylococcus aureus* pathogenesis. PLoS ONE 2008;3:e3198.
[14] Li M, Diep BA, Villaruz AE, Braughton KR, Jiang X, DeLeo FR, et al. Evolution of virulence in epidemic community-associated methicillin-resistant *Staphylococcus aureus*. Proc Natl Acad Sci U S A 2009;106:5883-8.