Panton-Valentine leukocidin positive *Staphylococcus aureus* infection in childhood: a case report

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Received: 26 January 2015, Accepted: 19 February 2015

SUMMARY: Karlı A, Belet N, Yanık K, Köken Ö, Kılıç M, Ceyhan-Bilgici M, Şensoy G. Panton-Valentine leukocidin positive *Staphylococcus aureus* infection in childhood: a case report. Turk J Pediatr 2015; 57: 615-617.

***Staphylococcus aureus* is a common cause of musculoskeletal infections. Panton-Valentine leukocidin (PVL) is an exotoxin produced by *S. aureus* and is an important virulence factor. PVL-positive *S. aureus* infections have been associated with soft tissue infections and necrotizing pneumonia. In this case report, the clinical course of a 13-year-old boy who was admitted with right hip pain and fever, diagnosed with a gluteal abscess and incidentally discovered multiple cavitary pulmonary lesions, and had a methicillin susceptible and PVL-producing *S. aureus* (MSSA) extracted from the abscess culture is presented. The related literature is also reviewed.**

Key words: Staphylococcus aureus, Panton-Valentine leukocidin, gluteal abscess, septic pulmonary embolus.

Staphylococcus aureus is the primary source of purulent infections around the world. The spectrum of staphylococcal infections ranges from mild superficial skin infections to lethal invasive infections. This bacterium causes most musculoskeletal infections in children. *S. aureus* strains have many virulence factors responsible for specific clinical situations. Panton-Valentine leukocidin (PVL) is an important virulence factor. Reports of PVL-producing *S. aureus* strains increased within the last decade[1-3]. The case of a 13-year-old child with a gluteal abscess caused by paronychia due to PVL-producing *S. aureus* is presented in this report.

Case Report

A 13-year-old boy was admitted with right hip pain and fever. The patient was previously healthy and consistent with his age, had developed paronychia in the big toe of his right foot but had not received any treatment. Physical examination revealed a body temperature of 38°C and sensitivity, rise in temperature, and erythema located in the right gluteal region. Even though the hip had free active-passive motion, hyperextension was painful, and desquamation was observed in the big toe of the right foot. The remaining system examinations were within normal limits. Laboratory values were as follows: hemoglobin 10.6 g/dl, white blood cell count 9,900/mm^3 (80% polymorphonuclear cells), platelets 420,000/mm^3, C-reactive protein 180 mg/L (normal 0-5 mg/L), and erythrocyte sedimentation rate 120 mm/h. Ultrasonography of the right gluteal region revealed a 50 × 33×16 mm thick, irregular abscess with thick septae. Cefotaxime 200 mg/kg/day treatment was initiated after blood cultures were obtained. Magnetic resonance imaging of the hip revealed a diffuse signal increase in the right iliac crest and increased fluid in the right hip joint. There was a 6×4.5 cm, irregular, multiloculated abscess in the gluteal region that showed peripheral contrasting, hypointensity in T1 and hyperintensity in T2 (Fig. 1). Since magnetic resonance imaging of the patient, whose chest radiography was normal, revealed multiple, dispersed lesions, some of which with cavitary nature, in the bilateral lower

regions of the lungs, a thoracic computed tomography scan was performed. Thorax tomography revealed multiple, dispersed cavitary lesions with air and fluid levels, 16×15 mm, located in the upper lobe of the right lung, consistent with septic pulmonary embolism (Fig. 2). Gluteal abscess drainage was performed on the patient who did not have tachypnea and auscultation findings of the lung. Methicillin-susceptible *S. aureus* (MSSA) was isolated from the culture obtained from the abscess. Blood culture was negative for growth. Cefazolin 100 mg/kg/day and clindamycin 40 mg/kg/day treatment was initiated on the 8th day of hospitalization. The fever had subsided on the 4th day of hospitalization; however, there was discharge from the abscess drainage point. Gluteal ultrasonography revealed a 33×15 mm dense abscess that was drained again from the same location. Since the *S. aureus* strains were found positive via polymerase chain reaction (PCR) technique for PVL and the abscess had reformed, cefazolin treatment was ceased on the 18th day, and linezolid (30 mg/kg/day) and clindamycin treatment was initiated. The immunologic tests were within normal limits. Lung tomography was performed on the 20th day of hospitalization. The patient was mostly cured except for a 10×8 mm cavitary lesion on the anterior upper lobe of the right lung and had a normal gluteal region. The patient was discharged with amoxicillin-clavulanate treatment on day 28. Antibiotherapy was stopped on the 10th post-discharge day. The 6-month follow-up was normal.

Discussion

Panton-Valentine leukocidin is a powerful virulence factor produced by *S. aureus* strains. It is an exotoxin that consists of two polypeptides known as LukS and LukF. PVL's role in invasive staphylococcal infections and pathogenesis is controversial. This toxin causes cell death by forming pores on the membranes of polymorphonuclear cells. PVL also causes the release of inflammatory mediators and loss of phagocyte functions. The release of mediators, in turn, causes local vasodilation, chemotaxis, neutrophil infiltration, production of superoxide ions, and tissue necrosis[3,4]. Studies performed on animal models revealed that PVL has a profound effect on staphylococcal infections concerning lung and bone tissue. However, the role of PVL in skin and soft tissue infections is subject to debate[5]. The incidence of MRSA infections is quite high in the United States but has a lower prevalence in Europe and Africa. The ratio of PVL-positive *S. aureus* changes due to geographic location, patient characteristics, localization of the infection, MRSA, and MSSA. According to different studies conducted in various countries, the incidence of PVL-positive MRSA strains varies between 74% and 100% while PVL-positive MSSA incidence is reported between 9% and 46%[1]. Studies conducted in Turkey report that the incidence of PVL-positive *S. aureus* isolated from the skin and soft tissue infections is 9.1%. All PVL-positive strains have been reported as MSSA[6]. Our case was community-acquired MSSA. A history of skin disease such as an abscess or a pustule, or nasopharyngeal existence of bacteria in the patient or people in contact with the patient can increase the risk of invasive *S. aureus* infections. Community-acquired PVL-positive *S. aureus* infections usually represent as furuncles,

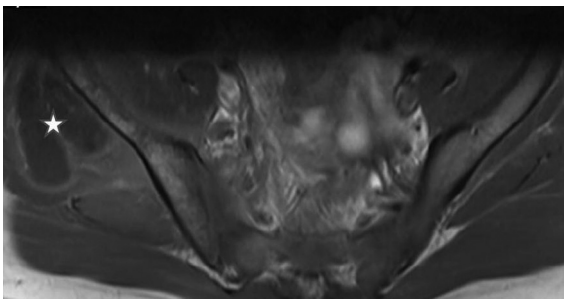

Fig. 1. Axial contrast-enhanced pelvic MRG section: Feature of compatible with existing abscess that lobulated contour and multioculated view, shows peripheral enhancement in right gluteal muscles.

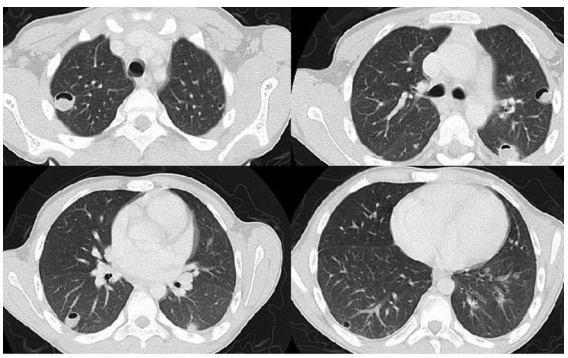

Fig. 2. Thoracic CT scans taken at different levels show cavitary lesions which are multiple, shows peripheral enhancement, contains air-fluid levels.

carbuncles, or cellulitis in patients and rarely cause invasive diseases[6,7]. PVL syndrome causes lethal soft tissue and bone infections, necrotizing pneumonia, deep vein thrombosis, sepsis, and multiple organ failure in otherwise healthy young adolescents and children[8]. Necrotizing pneumonia caused by PVL-positive *S. aureus* can be lethal in 75% of the cases. It can cause pulmonary abscess, cavitation, pleural effusion, and necrotizing pneumonia [9]. Bone infections caused by community-acquired *S. aureus* may cause secondary pulmonary involvement. Pulmonary findings are characteristically associated with septic pulmonary embolisms. Deep vein thrombosis can be diagnosed with Doppler ultrasonography. Septic pulmonary embolism can be observed as peripherally located, multiple, round cavitary lesions of different sizes with tomography. The underlying cause is often soft tissue infections[10]. We believe the possible source of *S. aureus* was the paronychia. We also believe that the gluteal abscess and the pulmonary septic embolus developed after the paronychia. However, we did not perform diagnostic tests for deep vein thrombosis. As a result of the antibiotherapy, the gluteal abscess disappeared, and a substantial recession was observed in the pulmonary embolus. Immediate initiation of aggressive antibiotherapy is important in lethal invasive infections such as sepsis, complicated musculoskeletal infections, or severe pneumonia. However, choice of an effective antibiotic is controversial. Clindamycin is the antibiotic of choice since it decreases toxin production in community-acquired PVL-positive MSSA. Cloxacillin and cefazolin can also be added to the treatment. Linezolid is used in MRSA-resistant strains. Some guidelines advise a combination of clindamycin and linezolid[10,11]. In this case, the initial cefazolin and clindamycin treatment was replaced with linezolid and clindamycin treatment due to the recurrence of the abscess. During the follow-up, the abscess did not reappear. Intravenous immunoglobulin (IVIG) can also be used for severe sepsis and pneumonia in PVL-positive cases [10,11]. We did not use IVIG since our case was not that severe.

Panton-Valentine leukocidin is an important virulence factor in *S. aureus* strains. It can cause fulminant invasive soft tissue infections in adolescents. Inflammatory markers, risk of complications, need for intensive care, and surgical interventions are higher and fever lasts longer in PVL-producing *S. aureus* strains. This case is presented to emphasize the importance of the clinical course and possible complications of PVL-producing *S. aureus* strains to clinicians.

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