International Journal of Infectious Diseases 26 (2014) 17–21

Contents lists available at ScienceDirect

International Journal of Infectious Diseases

journal homepage: www.elsevier.com/locate/ijid

Community-acquired necrotizing pneumonia caused by methicillin-resistant *Staphylococcus aureus* producing Panton–Valentine leukocidin in a Chinese teenager: case report and literature review

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ARTICLE INFO

Article history:
Received 11 January 2014
Received in revised form 22 February 2014
Accepted 28 February 2014

Corresponding Editor: Eskild Petersen, Aarhus, Denmark

Keywords:
Community-acquired methicillin-resistant *Staphylococcus aureus*
Community-acquired pneumonia
Panton–Valentine leukocidin
Linezolid
Teicoplanin
Fosfomycin

SUMMARY

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) has now been established as an important community-acquired pathogen. Although necrotizing pneumonia caused by community-acquired MRSA (CA-MRSA) strains producing Panton–Valentine leukocidin (PVL) has been reported with increasing frequency in many countries, it has been reported in only a few children younger than 1 year of age in Mainland China.
Methods: We describe a case of life-threatening necrotizing pneumonia due to PVL-positive CA-MRSA in a 15-year-old previously healthy female who presented with high fever, shivering, a dry cough, and dyspnea. Details of the clinical outcomes, microbiological data, and therapies for this patient were collected and compared with those of cases reported in the literature on CA-MRSA.
Results: Computed tomography (CT) findings showed cavitary consolidations in both lungs and bilateral pleural effusion. MRSA strains isolated from the patient's sputum and pleural fluid were susceptible to most non-β-lactam antimicrobial agents except for clindamycin and erythromycin. Both of these isolates tested positive for the *mec*A gene as well as PVL genes, and were identified as ST59-MRSA-SCC*mec* type IV-spa type t437. The patient was treated successfully with linezolid, fosfomycin, and teicoplanin.
Conclusions: To our knowledge, this is the first report from Mainland China of necrotizing pneumonia due to PVL-positive CA-MRSA among those aged older than 1 year. CA-MRSA necrotizing pneumonia should be considered in the differential diagnosis of severe community-acquired pneumonia, particularly in previously healthy individuals.

1. Introduction

Although traditionally regarded as a purely nosocomial pathogen, methicillin-resistant *Staphylococcus aureus* (MRSA) has now emerged as a community-acquired pathogen, affecting healthy people without recognized risk factors for MRSA acquisition.[1,2] In contrast to traditional healthcare-associated MRSA (HA-MRSA), these novel MRSA strains, termed community-acquired MRSA (CA-MRSA), carry smaller staphylococcal chromosomal cassette *mec* (SCC*mec*) elements (type IV or type V) and retain susceptibility to a wider range of antimicrobial agents, except for β-lactams.[1–4] Additionally, CA-MRSA strains are more likely than HA-MRSA strains to carry the Panton–Valentine leukocidin (PVL), a strong cytolytic factor with a unique ability to create pores in the cell membranes of human neutrophils and induce the release of chemotactic factors, although its role in the pathogenesis of CA-MRSA infections is controversial and PVL-negative CA-MRSA clones have been reported worldwide in recent years.[1,2,5,6]

CA-MRSA is primarily associated with skin and soft tissue infections (SSTIs) in previously healthy individuals;[2,7] however, it may occasionally cause invasive and severe infections like necrotizing pneumonia, necrotizing fasciitis, pyomyositis, osteomyelitis, and sepsis.[1,6] As one of the most severe illnesses caused

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http://dx.doi.org/10.1016/j.ijid.2014.02.025

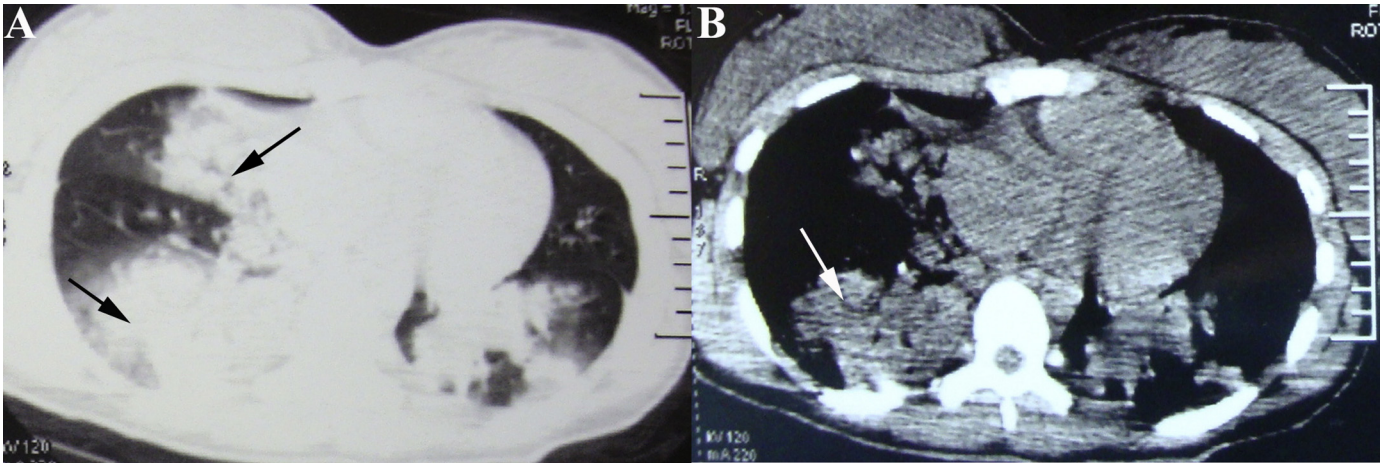

Figure 1. CT images at 5 days after the onset of pneumonia, showing irregular consolidations with interspersed small lucent areas in both lungs: A, black arrows; B, white arrow.

by CA-MRSA strains producing PVL, necrotizing pneumonia has been reported with increasing frequency in many countries and has a high mortality rate of 56–63%.[1,8] In China, data regarding CA-MRSA are limited and necrotizing pneumonia caused by PVL-positive CA-MRSA has been reported in only a few infants.[9]

Herein, we present a case of life-threatening pneumonia caused by CA-MRSA with a favorable outcome in a 15-year-old previously healthy teenage girl. To the best of our knowledge, this is the first report from Mainland China of necrotizing pneumonia due to PVL-positive CA-MRSA among those aged older than 1 year. We also review the literature related to CA-MRSA pneumonia.

2. Case report

A 15-year-old previously healthy female presented to the Chinese People's Liberation Army General Hospital with a 5-day history of high fever, sore throat, and a dry cough, as well as a 1-day history of shivering and dyspnea. She had been diagnosed with community-acquired pneumonia (CAP) and treated with intravenous azithromycin in a local hospital for the first 4 days. However, her clinical condition deteriorated rapidly. On the fifth day, she developed dyspnea requiring oxygen support, accompanied by shivering and a high fever of 41 °C. A chest computed tomography (CT) scan showed irregular consolidations with interspersed small lucent areas in both lungs (Figure 1).

Her vital signs on admission revealed a temperature of 39.9 °C, heart rate of 102 beats/min, and blood pressure of 140/85 mmHg. Physical examination showed pharyngeal hyperemia and swelling of the tonsils. On chest auscultation, lower respiratory sounds and a few moist rales were heard in the base areas of both lungs. Initial blood investigations revealed a white blood cell count (WBC) of 10.1×10^9/l with 94.7% neutrophils and normal hemoglobin and platelets. Arterial blood gas analysis showed a pH of 7.411, a partial pressure of carbon dioxide ($PaCO_2$) of 32.9 mmHg, and a partial pressure of oxygen, arterial (PaO_2) of 84.4 mmHg at 9 l/min inspired oxygen with a partial rebreathing mask. Serum C-reactive protein (CRP) and procalcitonin (PCT) were both markedly elevated at 14.2 mg/dl and 26.68 ng/ml, respectively. A PCR test performed on a nasal swab was negative for influenza virus. The patient underwent drainage of the pleural cavity and received a preliminary diagnosis of empyema. Pleural fluid examination revealed yellowish purulent features: WBC 2.88×10^9/l with 90% neutrophils, lactate dehydrogenase (LDH) 1302.2 IU/l, total protein (TP) 39.1 g/l, adenosine deaminase (ADA) 33.7 IU/l, glucose 2.4 mmol/l, and chloride 106 mmol/l.

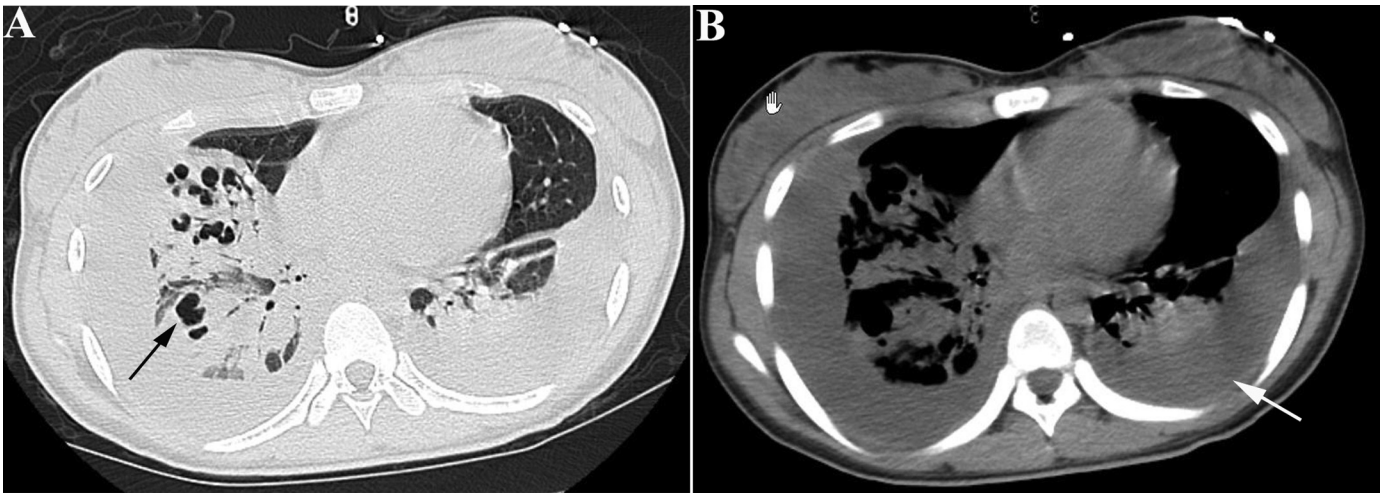

Figure 2. CT images at 8 days after the onset of pneumonia, showing multiple cavitary lesions in both lungs (A, black arrow) and bilateral pleural effusion (B, white arrow).