Community-Acquired Methicillin Resistant *Staphylococcus* aureus Bacteremia: Case Series

Veni E JK,¹ Bhat G,¹ Shalini S M,¹ Kumar P,² Chakrapani M,³ Baliga S¹

¹Department of Microbiology

²Department of Dermatology, Venereology and Leprosy

³Department of Internal Medicine

Kasturba Medical College (Manipal University)

Mangalore, India

Corresponding Author

Gopalkrishna Bhat

Kasturba Medical College (Manipal University)

Mangalore, India

Email: gopalkrishna.bhat@manipal.edu

Citation

Veni E JK, Gopalkrishna Bhat G, Shalini S M, Kumar P, Chakrapani M, Baliga S. Community-Acquired Methicillin Resistant *Staphylococcus aureus* Bacteremia: Case Series. *Kathmandu Univ Med J* 2015;49(1):77-9.

ABSTRACT

Community-acquired methicillin resistant *Staphylococcus aureus* (MRSA) usually causes skin and soft tissue infections. However, community-acquired methicillin resistant *S.aureus* has been identified as a causative agent of many invasive infections like necrotizing fasciitis, pneumonia and bacteremia. Risk factors such as immunodeficiency and skin and soft tissue infections have been identified for acquiring bacteremia. We present four cases of bacteremia caused by community-acquired methicillin resistant *S.aureus*, risk factors and outcome.

KEY WORDS

Bacteremia, community acquired, MRSA, mortality, risk factors

INTRODUCTION

Community-acquired methicillin resistant *Staphylococcus aureus* (CA-MRSA) has emerged as a significant pathogen since 1990s. It mostly causes skin and soft tissue infections (SSTI) in young healthy individuals without any health-care associated risk factors. But, recently CA-MRSA has been reported to cause invasive infections like bacteremia, necrotising pneumonia and osteomyelitis. However, limited data is available regarding invasive infections caused by CA-MRSA in developing countries. CA-MRSA is emerging as a pathogen responsible for bacteremia. ACA-MRSA SSTI and intravenous drug use are some of the risk factors for CA-MRSA bacteremia. CA-MRSA bacteremia is associated with prolonged hospital stay, endocarditis, septicaemia and death.

CASE-REPORT

Case 1

A three-year-old girl presented with severe pain over right hip and knee with inability to move since one week. On preliminary examination, the patient was febrile (temperature – 39.5°C) with blood pressure- 98/60 mmHg, pulse rate- 92 bpm and respiratory rate- 28 breaths/ minute. Laboratory data showed haemoglobin- 9.6 g/ dL, total leukocyte count- 2900 cells/mm3 with 79.5% neutrophils, 17.7% lymphocytes, 1.0% basophils and 2.7% monocytes; erythrocyte sedimentation rate-75 mm/1hour and C-reactive protein- 270 mg/dL. Ultrasound examination of right knee showed minimal periarticular soft tissue edema and right hip showed minimal hip joint effusion. Also multiple discrete fluid filled, erythematous, vesicular lesions were seen all over the body. The patient was diagnosed with right knee and hip septic arthritis and bullous impetigo with erythema multiforme. Empirical treatment was started with cefepime and clindamycin. Pus swab from skin lesion and blood were collected for culture on the day of admission prior to antibiotic therapy. Both specimens grew S.aureus which was identified as MRSA by standard methods.8,9 The isolates from pus and blood had the same antibiotic susceptibility profile showing resistance to amoxicillin/clavulanic acid and penicillin while susceptible to ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin, linezolid, rifampicin, teicoplanin and tetracycline. The isolates were considered to be community-acquired based on Centre for Disease Control and Prevention (CDC) guidelines, ¹⁰ where the organism was isolated within 48 hours of hospitalization; the patient did not have any history of MRSA infection; no medical history in the past year of hospitalization, admission to a nursing home, skilled nursing facility or hospice, dialysis and surgery; no permanent indwelling catheters or medical devices that pass through the skin into the body. Treatment was changed to vancomycin and rifampicin following blood culture and sensitivity results. The general condition of the patient improved and was discharged after 12 days of treatment.

Case 2

A 61-year-old man presented with fever; itchy, fluid filled lesions all over the body and pain with discharge from eyes since eight days. Vital data showed blood pressure- 128/70 mmHg, pulse rate- 106 bpm, respiratory rate- 20 breaths/ minute and temperature- 39° C. Laboratory investigations showed haemoglobin-13.8 g/dL, total leukocyte count-6000 cells/mm3 with neutrophils-66%, lymphocytes-24%, basophils-1%, monocytes-10%; erythrocyte sedimentation rate- 31 mm/1hour. Physical examination showed multiple pleomorphic lesions- vesicles, pustules, crusted lesions all over the body. Conjunctiva was congested with purulent discharge. The patient was diagnosed with varicella infection. Fluid from lesions and blood were collected for culture on the day of admission. Empirical treatment was started with valacyclovir, cefotaxime and clindamycin. Culture of fluid from lesion and blood grew S.aureus which was identified as MRSA using standard methods.^{8,9} MRSA from both specimens were resistant to amoxicillin/clavulanic acid, erythromycin and penicillin while susceptible to ciprofloxacin, clindamycin, cotrimoxazole, gentamicin, linezolid, rifampicin, teicoplanin and tetracycline. Since MRSA was resistant to erythromycin and susceptible to clindamycin, D-test was done to know if it had inducible clindamycin resistance. 11 The D-test was positive, indicating inducible clindamycin resistance. MRSA was identified as community-acquired based on CDC guidelines.¹⁰ Treatment was changed to teicoplanin. The general condition of the patient improved and was discharged after four days of treatment.

Case 3

A 34-year-old woman, a known case of pemphigus vulgaris presented with chief complaints of increased lesions, fever and malaise since 10 days. She was diagnosed with pemphigus vulgaris in 2005 and was on treatment with immunosuppressant drugs. Physical examination showed multiple crusted lesions with few flaccid bullae on face, upper chest, hands and thighs. On preliminary examination, vital data showed blood pressure- 110/70

mmHg, pulse rate- 110 bpm, respiratory rate- 30 breaths/ minute and temperature- 39° C. Laboratory data showed haemoglobin- 11.5 g/dL, total leukocyte count- 500 cells/mm3 with neutrophils- 62%, lymphocytes- 31%, monocytes- 7%, basophils- 1%; erythrocyte sedimentation rate- 31 mm/1hour. On admission, pus from lesions and blood were collected for culture and sensitivity. Due to low total leukocyte count, immunosuppressant was stopped and treatment for low total leukocyte count was started with filgrastim along with cefotaxime. S.aureus grew from both blood and pus specimen from lesions.8 S.aureus from blood culture was identified as MRSA using standard procedures.9 It was resistant to amoxicillin/clavulanic acid and penicillin and susceptible to ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin, linezolid, rifampicin, teicoplanin and tetracycline. CDC guidelines,¹⁰ were used for identifying MRSA as community-acquired. Vancomycin was included for treatment following blood culture report. Total leukocyte count began to increase steadily up to 2100 cells/mm3 till day 13 when the condition of the patient improved. However on day 14, patient developed breathlessness and total leukocyte count dropped to 1300 cells/mm3. Patient was shifted to the intensive care unit and was continuously monitored. She died after 11 days of treatment, primary cause of death being septicaemia and secondary causes were febrile neutropenia and pemphigus vulgaris.

Case 4

A 52-year-old man, known case of type 2 diabetes mellitus, presented to the emergency department complaining of fever and three to four episodes of hemoptysis per day since seven days. Fever was moderate to high grade associated with chills and rigor. On preliminary examination, vital data showed blood pressure- 130/70 mmHg, pulse rate- 76 bpm, respiratory rate- 26 breaths/minute and temperature-38° C. Laboratory investigation showed haemoglobin-9.2 g/dL, total leukocyte count- 4900 cells/mm3 with neutrophils-72.4%, lymphocytes- 20%, monocytes- 3.4% and basophils- 0.5%; erythrocyte sedimentation rate- 25 mm/1hour. Respiratory system examination showed left basal crepts; chest X-ray revealed homogenous opacity in the left lower lobe; bronchoscopy showed left lower lobe bleed. The patient was diagnosed with pneumonia. Patient was treated empirically with augmentin. Sputum and blood were collected for culture on the day of admission prior to therapy. Blood culture grew S.aureus which was further identified as MRSA based on standard procedures.^{8,9} MRSA was resistant to amoxicillin/clavulanic acid and penicillin and susceptible to ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin, linezolid, rifampicin, teicoplanin and tetracycline. It was identified as community-acquired based on CDC guidelines.¹⁰ Clindamycin was started following blood culture result. The general condition of the patient improved and was discharged after three days of treatment.

DISCUSSION

The present study shows the significance of CA-MRSA as a causative agent of bacteremia. Increased rate of CA-MRSA SSTI may have resulted in an increase in the rate of bacteremia in the community. 1,3,5,12 Other clinical features associated with CA-MRSA bacteremia are bone and joint infections, chronic liver disease, chronic obstructive pulmonary disease, deep abscesses, diabetes mellitus, endocarditis, necrotizing pneumonia, renal insufficiency and septic shock. 1,4,5,12,13 Intravenous drug use and homelessness can be significant risk factors for CA-MRSA bacteremia. A previous study has reported significant association of CA-MRSA with a high rate of endocarditis among HIV patients with bacteremia. However, CA-MRSA bacteremia has also been reported in patients with no risk factors.

All MRSA isolates from the cases present here were susceptible to most non-beta lactam antibiotics, a phenotypic property of CA-MRSA.¹⁰ In the present study,

all the four patients presented with at least one risk factor for CA-MRSA bacteremia. Cases one, two and three had SSTI, out of which MRSA was isolated from both pus and blood in two patients. Both isolates from blood and pus in two patients had the same antibiotic susceptibility profile indicating that MRSA from SSTI may have gained entry into the blood stream. Case four had type II diabetes mellitus, one of the risk factors for bacteremia. CA-MRSA bacteremia can lead to high risk of mortality, increased duration of hospital stay and risk of recurrent or persistent infection. Some factors associated with increased mortality are age, alcoholism, chronic liver disease, pneumonia, septic shock and immunosuppression. In the present study, death of one immunosuppressed patient was due to CA-MRSA bacteremia.

In conclusion, early diagnosis of CA-MRSA bacteremia and antibiotic susceptibility profile determination will help start appropriate antibiotic treatment and management of the case.

REFERENCES

- Kempker RR, Farley MM, Ladson JL, Satola S, Ray SM. Association of methicillin-resistant Staphylococcus aureus (MRSA) USA300 genotype with mortality in MRSA bacteremia. J Infect. 2010 Nov;61(5):372-81.
- Klevens RM, Morrison MA, Nadle J, Petit S, Gershman K, Ray S et al. Active Bacterial Core surveillance (ABCs) MRSA Investigators. Invasive methicillin-resistant Staphylococcus aureus infections in the United States. JAMA. 2007 Oct 17;298(15):1763-71.
- Tattevin P, Schwartz BS, Graber CJ, Volinski J, Bhukhen A, Bhukhen A et al. Concurrent epidemics of skin and soft tissue infection and bloodstream infection due to community-associated methicillinresistant Staphylococcus aureus. Clin Infect Dis. 2012 Sep;55(6): 781-8.
- 4. Chi CY, Wong WW, Fung CP, Yu KW, Liu CY. Epidemiology of community-acquired Staphylococcus aureus bacteremia. *J Microbiol Immunol Infect*. 2004 Feb; 37(1):16-23.
- Murray RJ, Lim TT, Pearson JC, Grubb WB, Lum GD. Community-onset methicillin-resistant Staphylococcus aureus bacteremia in Northern Australia. Int J Infect Dis. 2004 Sep;8(5):275-83.
- Centers for Disease Control and Prevention. Four pediatric deaths from community-acquired methicillin-resistant Staphylococcus aureus-Minnesota and North Dakota, 1997-1999. JAMA. 1999 Sep 22-29;282(12):1123-5.
- Furuno JP, Johnson JK, Schweizer ML, Uche A, Stine OC, Shurland SM et al. Community-associated methicillin-resistant Staphylococcus aureus bacteremia and endocarditis among HIV patients: a cohort study. BMC Infect Dis. 2011 Oct 31;11:298-304.

- Winn Jr WC, Allen SD, Janda WM, Koneman E, Procop G, Schreckenberger PC, et al. Gram positive cocci: Part I Staphylococci and related Gram positive cocci. Koneman's Color Atlas and Textbook of Diagnostic Microbiology. 6th ed. Philadelphia: Lippincott; 2006.
- Clinical and Laboratory Standards Institute. Performance standards for antimicrobial Disc diffusion tests. CLSI document M100-S232. CLSI 2013.
- David MZ, Daum RS. Community-associated methicillin-resistant Staphylococcus aureus: epidemiology and clinical consequences of an emerging epidemic. Clin Microbiol Rev. 2010 Jul;23(3):616-87.
- Fiebelkorn KR, Crawford SA, McElmeel ML, Jorgensen JH. Practical disk diffusion method for detection of inducible clindamycin resistance in Staphylococcus aureus and Coagulase-Negative Staphylococci. *J Clin Microbiol*. 2003 Oct;41(10):4740–4.
- Wang JL, Chen SY, Wang JT, Wu GH, Chiang WC, Hsueh PR et al. Comparison of both clinical features and mortality risk associated with bacteremia due to community-acquired methicillin-resistant Staphylococcus aureus and methicillin-susceptible S. aureus. Clin Infect Dis. 2008 Mar 15;46(6):799-806.
- Lee JY, Chong YP, Kim T, Hong HL, Park SJ, Lee ES et al. Bone and joint infection as a predictor of community-acquired methicillin-resistant Staphylococcus aureus bacteraemia: a comparative cohort study. J Antimicrob Chemother. 2014 Jul;69(7):1966-71.