

## Boil to Sepsis: Case of Community Acquired MRSA

Under-recognition and under-reporting marks the epidemiological profile of CA-MRSA (community acquired methicillin resistant *Staphylococcus aureus*) in India. We present the first case report with Panton-Valentine leukocidin (PVL) gene isolation.

The index case, a 13 year male, previously well, developed a boil in left elbow followed by fever, rapidly progressive tender swelling of left leg, respiratory distress and septic shock within next 12 hours warranting intubation, mechanical ventilation and use of pressors. Thrombotic and vasculitis work-up, coagulation, hematological, metabolic parameters were normal with polymorphonuclear leucocytosis. Limb Doppler showed femoropopliteal deep venous thrombosis (DVT). CT chest revealed patchy consolidation in both lungs CT limb and echocardiogram were normal. Blood culture sent on day 1 of illness grew MRSA.

The patient was started on linezolid and low molecular weight heparin. Despite 7 days of IV linezolid therapy, blood cultures remained positive for MRSA. We switched to clindamycin (initially intravenous and later oral), given for a total of 4 weeks. Genetic studies confirmed presence of PVL gene.

CA-MRSA infections differ from hospital acquired MRSA by predominantly presenting as minor skin and soft tissue infections in risk free healthy hosts(1). Infection often carries PVL toxin that kills leucocytes and is associated with severe course of disease(1,2). Our patient had DVT, a well recognized association in patients with CA-MRSA. "PVL syndrome" includes osteomyelitis, skin infections, pneumonia and DVT(3).

The recommended antibiotic therapy for severe infections with CA-MRSA include vancomycin,

linezolid and clindamycin(2,4,5). Vancomycin is used to treat sensitive (MIC <1µg/mL) strains. Data of resistance to this drug in India is lacking. Vancomycin MIC was of intermediate range here (2-4 µg/mL) and hence was not prescribed. Linezolid (IV or oral) is recommended as standard ICU therapy for suspected CA-MRSA pneumonia due to good lung penetration.

*Clindamycin (IV and oral)*

2.4% to 10% of CA-MRSA isolates initially reported susceptible to clindamycin (but resistant to erythromycin) may develop clindamycin resistance (detected by the D-zone disk diffusion test) resulting in treatment failure. Our patient's D test was negative. Unlike vancomycin, linezolid and clindamycin have excellent anti-toxin activity.

The PVL toxin and CA-MRSA are under-recognized entities in India. In very sick patients with risk factors for MRSA, possibility of CA-MRSA infection must be entertained and vancomycin/clindamycin empirically used with de-escalation later. However, it is vital to avoid indiscriminate misuse of higher and antibiotics in the management of methicillin sensitive staphylococcal infections where cloxacillin is the drug of choice.

### ACKNOWLEDGMENTS

Kerry Williams, Clinical Scientist, Royal Free Hospital London isolated the PVL gene from the strain; Dr Abdul Gafur for helping us send the strain for genetic analysis.

**S Gayathri and J Indira**

*Pediatric Intensive Care Unit,  
Apollo Hospitals (Main), Greams Road,  
Chennai 600 006, India.  
E-mail: gayathri.subramanian@gmail.com*

### REFERENCES

1. Naimi TS, LeDell KH, Como-Sabetti K, Borchardt SM, Boxrud DJ, Etienne J, *et al.* Comparison of community and health care-associated methicillin-

- resistant *Staphylococcus aureus* infection. *JAMA* 2003; 290: 2976-2984.
2. Thomas RW, Gene H, Bradley WF. Community-associated methicillin-resistant *Staphylococcus aureus*. *Emerg Med Clin N Am* 2008; 26: 431-455.
  3. Swaminathan A, Massasso D, Gotis-Graham I, Gosbell I. Fulminant methicillin-sensitive *Staphylococcus aureus* infection in a healthy adolescent, highlighting 'Panton-Valentine leucocidin syndrome'. *Intern Med J* 2006; 36: 744-747.
  4. Gorwitz RJ, Jernigan DB, Powers JH, Jernigan JA, and Participants in the CDC convened experts meeting on management of MRSA in the community. Strategies for clinical management of MRSA in the community: Summary of an experts' meeting convened by the Centers for Disease Control and Prevention. Atlanta: CDC; 2006.
  5. Mandell LA, Wunderink RG, Anzueto A, Bartlett JG, Campbell GD, Dean NC, *et al.* Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults. *Clin Infect Dis* 2007; 44 (Suppl 2): 827-872.

## Post Varicella Thrombosis

We report a case of extensive thrombosis in a 12 year old boy, who had varicella 15 days earlier. The child presented with headache and generalized tonic clonic seizures for 2 hours. On examination, he had healed scars of varicella. Neurological examination revealed a Glasgow Coma Score of 9/15. CSF analysis, complete blood counts, RFT, LFT, blood sugar, serum electro-lytes, PT and APTT were normal. CT Brain revealed multiple areas of hemorrhage with perilesional edema involving bilateral parietal and left frontal region. The child was started on anticonvulsants. Magnetic resonance venography of brain revealed superior sagittal and bilateral transverse sinus thrombosis. Prothrombotic screen (Protein S, Protein C levels, Factor V Leiden Mutation, Anti-thrombin III levels and anti - cardiolipin antibodies) was planned, but could not be done due to financial constraints.

The child was started on LMW heparin along with oral anticoagulants. The child's sensorium improved gradually over 5 days. On day five of admission, he developed left leg pain with swelling. Doppler study revealed extensive thrombosis of left external iliac, femoral and popliteal veins. He improved with limb elevation and analgesics. He was discharged on anticonvulsants and oral anticoagulants maintaining an INR of 2-3. On follow up there are no neurologic sequelae or subsequent

episodes of thrombosis. Fresh frozen plasma was not used in child as the response to the above treatment was satisfactory.

The incidence of serious complications after varicella infection is 8.5/1 lakh population(1). Thrombotic complications are known especially involving the cerebral vasculature(2). Eidelberg, *et al.*(3) suggested that virus mediated endothelial injury promotes local thrombosis but transient deficiency of protein S activity (due to induction of anti-protein S auto antibodies) is also a causal factor(4,5). These antibodies persist for only a few months. The frequency with which antibodies to proteins S are induced in children during varicella infection is unknown. Thrombosis is more common in individuals with Factor V Leiden, which is a factor V variant resulting from a single point mutation. It increases the risk for thrombosis as it confers resistance to activated protein C [5]. Prognosis in post varicella thrombosis is good. A prothrombotic screen after recovery, to diagnose hereditary prothrombotic states that need life long anticoagulants, is advisable.

**Padmasani Venkat Ramanan  
and Kanav Anand**

*Department of Pediatrics,  
Sri Ramachandra Medical College,  
Porur, Chennai 600 116, India.*

### REFERENCES

1. Ziebold C, Von Kries R, Lang R, Weigl J, and HJ.