

CASE REPORT

Methicillin resistant *Staphylococcus aureus* meningitis

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Abstract

Methicillin resistant *Staphylococcus aureus* (MRSA) meningitis is rarely known to occur in children. We report an 11-year-old girl with fever, headache and vomiting, right hemiparesis with left-sided upper motor neuron facial nerve palsy and bladder incontinence. On investigation, she was found to have MRSA meningitis with an acute left thalamo-capsular infarct. She was treated with vancomycin, linezolid and rifampicin. She recovered successfully with residual right-sided lower limb monoparesis. MRSA meningitis is rare but can occur in children.

INTRODUCTION

Community-acquired (CA) methicillin resistant *Staphylococcus aureus* (MRSA) has emerged as a pathogen of major importance in pediatric patients. Though hospital-acquired (HA) MRSA is commonly seen in hospitalized individuals with underlying predisposing medical conditions, CA-MRSA may occur without any predisposing factor and usually presents with skin and soft tissue infection [1]. Common organisms causing meningitis in neonates and children are Group B *Streptococcus*, *Escherichia coli*, *Listeria monocytogenes*, *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Neisseria meningitidis* [2]. In one study, incidence of *S. aureus* meningitis was 6% of which MRSA meningitis was 18% [3]. MRSA meningitis is seen equally in post-operative patients and hematogenous meningitis [4]. We present an 11-year-old girl with CA-MRSA with meningitis who had a favorable outcome.

CASE REPORT

An 11-year-old girl presented with fever for 10 days, headache for 4 days and vomiting for 2 days, altered sensorium and left eye swelling since the morning of presentation to us. On examination, weight was 23 kg and height was 143 cm. She had neck stiffness with right-sided hemiparesis with left-sided upper motor neuron facial nerve palsy and bladder incontinence.

Deep tendon reflexes were brisk bilaterally. She was drowsy but arousable. Other systems were normal. Left eye cellulitis was present. She had received intramuscular ceftriaxone in left buttock for the above complaints from another hospital. There was no history of boils or pus-filled vesicles previously.

Investigations showed hemoglobin 9.3g%, white blood cells count 13 600 cells/cumm (92% polymorphs, 8% lymphocytes), platelets 3 59 000/cumm, erythrocyte sedimentation rate 93 mm at the end of 1 h, C-reactive protein 61 mg/l and blood culture showed no growth. Serial cerebrospinal fluid (CSF) examinations were done and are depicted in Table I. CSF culture grew MRSA on Day 11 of hospitalization sensitive to vancomycin, linezolid and rifampicin. CSF latex agglutination for *S. pneumoniae* and *H. influenzae* was negative. CSF TB-MGIT and Gene X pert were negative. MRI Brain with contrast (Fig. 1) revealed an acute infarct in the left thalamo-capsular region with a discrete focus of acute infarct in the left subcortical frontal white matter, appearing hypointense on ADC maps and hyperintense on FLAIR images. Mild diffuse abnormal leptomeningeal enhancement was also noted suggestive of meningitis.

She was initially treated with IV ceftriaxone and IV vancomycin for meningitis but had no response. After CSF grew MRSA, her antibiotics were changed to IV vancomycin (60 mg/kg/day q6 hourly), IV linezolid (10 mg/kg/dose thrice daily) and rifampicin (10 mg/kg/dose twice daily) for 28 days. Echocardiography did

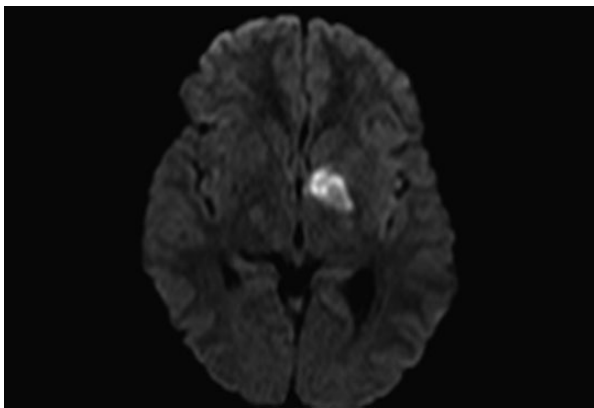
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Table 1. Cerebrospinal fluid examination

	Day 1	Day 6	Day 11
Appearance	Colorless, clear	Colorless, hazy	Colorless, slightly hazy
Total cell count/cumm	2110	1180	3200
Polymorphs (%)	90	30	50
Lymphocytes (%)	10	70	50
Proteins (mgm%)	550	65	60
Sugar (mgm%)	24	44	32
Serum glucose (mgm%)	86	94	-
Gram stain	Pus cells, occasional red blood cells, occasional Gram negative cocci	Pus cells, no organism seen	Pus cells, no organism seen
India ink			Cryptococcus not seen
Zeil-Neilson stain	Acid fast bacilli (AFB) not seen	AFB not seen	AFB not seen

**Figure 1:** MRI Brain with contrast (axial view) showing acute left thalamo-capsular infarct.

not show infective endocarditis. Decolonization measures with chlorhexidine bath and nasal mupirocin were done daily. Nasal swab of patient and her parents showed no growth.

The child improved. Right-sided lower limb paresis persisted. Facial nerve palsy resolved, and bladder continence improved. Child is now on regular follow-up.

DISCUSSION

Central nervous system (CNS) infections caused by MRSA occur as a complication of a neurosurgical procedure, in association with a contiguous focus of infection, or hematogenously as a complication of bacteremia or infective endocarditis. Treatment is difficult because of the critical location of these infections and the blood brain barrier, which limits the penetration of systemically administered antibiotics to the site of infection. Thus, surgical drainage of focal abscesses and removal of any foreign body, such as an infected ventriculo-peritoneal shunt, should be performed whenever possible [5].

Children identified to have Panton-Valentine leukocidin (PVL) CA-MRSA infections have higher levels of inflammation markers (C-reactive protein and sedimentation rate), and it has been linked to severe necrotizing pneumonia, furunculosis and severe osteomyelitis. Acquisition of Type I arginine catabolic mobile element (ACME) may allow CA-MRSA to colonize the skin on a permanent basis, thereby enhancing the likelihood of a skin infection occurring upon any disruption of the skin barrier. Phenol-soluble modulins (PSM) peptides are expressed at a higher

level in CA-MRSA when compared with HA-MRSA, which prompts suggestion that differences in global virulence regulation could be an important factor in CA-MRSA virulence [6].

Two distinct pathogenic mechanisms for MRSA meningitis include post-operative and spontaneous [7]. Other predisposing factors for CA-MRSA include preseptal cellulitis, otitis media and clivial osteomyelitis [8, 9]. In our patient, left eye cellulitis was a predisposing factor for spontaneous MRSA meningitis.

Other complications of CA-MRSA include zygomatic osteomyelitis, cavernous sinus thrombosis and Bell's palsy [8, 10]. In our patient, an acute infarct in the left thalamo-capsular region was seen as a complication with residual right lower limb monoparesis seen as a focal neurological deficit.

Vancomycin and rifampicin are the antibiotics of choice for MRSA meningitis with complications as per Infectious Disease Society of America guidelines and are given for 4–6 weeks. Vancomycin has been the drug of choice, but outcomes have been very poor when it has been used as monotherapy. Experts recommend rifampicin in combination with vancomycin for meningitis and other CNS infections. High dose continuous infusion of vancomycin may be considered in patients not responding to standard dosing methods. CSF penetration was increased, and concentrations were almost doubled, compared with standard dosing, when vancomycin was administered as a 15 mg/kg loading dose, followed by continuous infusion of 50–60 mg/kg/day for patients with normal renal function [5]. Similarly in our patient, there was initially no response to vancomycin, and after culture grew MRSA, linezolid and rifampicin were added following which patient responded.

CA-MRSA meningitis is rare in children and can occur without predisposing factors. A strong clinical suspicion and timely intervention are required to ensure good outcome.

AUTHORS' CONTRIBUTIONS

All authors were involved in patient management, preparation of manuscript and approving the final version.

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CONFLICT OF INTEREST STATEMENT

None declared.

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