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Community-acquired Methicillin-resistant *Staphylococcus aureus* (CA-MRSA) Infection

Methicillin resistant strains of *Staphylococcus aureus* have been recognized for over thirty years as endemic pathogens in virtually all hospitals and healthcare facilities, causing nosocomial infection and colonization. MRSA infections are also a known complication of injecting drug use.

Over the past few years in the U.S., increasing numbers of MRSA infections, termed *community-acquired*, have been described among persons without the following risk factors for MRSA infection:

- History of past MRSA infection or colonization
- History in the past year of:
 - ✓ Hospitalization
 - ✓ Admission to a nursing home, skilled nursing facility, or hospice
 - ✓ Dialysis
 - ✓ Surgery
- Presence of permanent indwelling catheters or medical devices that pass through the skin into the body.

Public Health has received sporadic reports of CA-MRSA, since February 2003, when Public Health requested that clinicians, hospital epidemiologists, and microbiology laboratory personnel report increases in, or unusual clinical manifestations of CA-MRSA infections.

The following report describes a recent case of CA-MRSA infection in King County:

In summer 2003, a college student presented to the emergency department with a five-day history of low back pain of sudden onset, followed by developing numbness in the perineum, lower extremity weakness, and inability to urinate or defecate. Two weeks prior to symptom onset, he noticed a pustule on his right thigh; a second lesion appeared on the right buttocks several days later. He had no recognized risk factors for MRSA infection and no history of injection drug use.

On examination, vital signs indicated a temperature of 102.6 degrees F, blood pressure 100/70, pulse 140 per minute, respiration 26 per minute. Cardiac examination was normal. A single 2-centimeter furuncle was present on the right buttock without fluctuance. Neurologic exam confirmed bilateral lower extremity paresis, neurogenic bladder, absent anal reflex, and perineal hypesthesia. There were no manifestations of injection drug use. Laboratory data showed a white blood cell count of 11,500/mm³ with left shift, and serum creatine kinase of 2050 IU/ml. Human immunodeficiency virus antibody was negative. He was

presumptively diagnosed with bacterial sepsis from a cutaneous source, possibly complicated by epidural abscess.

He was initially treated with vancomycin and ceftriaxone. Within 12 hours of admission, blood culture yielded gram-positive cocci, and cefazolin was substituted for ceftriaxone. An MRI of the back revealed inflammation and micro abscesses of the lumbar paravertebral muscles and lumbar nerve root compression. Inflammation from the lower cervical to lumbar regions were demonstrated, but no gross abscess or evidence vertebral osteomyelitis was noted. Blood cultures confirmed *Staphylococcus aureus* resistant to penicillin, oxacillin, erythromycin and cefazolin (methicillin resistant *Staphylococcus aureus* or MRSA). The minimum inhibitory and bacteriocidal concentrations for vancomycin were 1.0 mcg/ml, and 4.0 mcg/ml, respectively. Despite the absence of drainable soft tissue abscess, valvular vegetations, or other identifiable sites of active suppuration, blood cultures remained persistently positive for MRSA over the ensuing six days. He developed persistent hypotension, acute respiratory failure, progressive pulmonary barotrauma and pneumothorax. An abdominal ultrasound revealed an inferior vena caval thrombus on the last day of life. He expired on the seventh hospital day.

Post mortem examination revealed diffuse severe bilateral pneumonia with multiple micro abscesses. A thirteen centimeter thrombus extended from the inferior vena cava to the external and internal iliac veins. There was no evidence of endocarditis. Tissue gram stain of post mortem lung tissue, inferior vena cava thrombus, and lumbar epidural tissue revealed large numbers of intracellular gram positive cocci consistent with *Staphylococci*.

MRSA is not a legally notifiable condition, therefore the incidence of CA-MRSA infections is not known. However, the infection control program at Group Health Cooperative has been monitoring the number of "first isolates" of MRSA from their entire

enrollee population, because individuals with "first isolates" may represent new acquisitions of MRSA. Since 1999, there has been a four-fold increase in the absolute number of individuals with newly acquired MRSA, and the proportion of individuals with newly acquired MRSA, who have never been inpatients in an acute care hospital, skilled nursing facility, or long term care institution, has increased from 23% in 1999, to 44% in 2003.

In response to the emergence of CA-MRSA:

- Health care providers should consider MRSA infection when evaluating and treating persons with community-acquired skin and soft tissue infections.
- Empiric antibiotic treatment for serious or potentially life-threatening community-acquired *Staphylococcus aureus* infection should include an agent with activity against methicillin-resistant strains until results of culture and susceptibility testing are available.

For more information about CA-MRSA, see:
www.cdc.gov/ncidod/hip/Aresist/mrsa.htm

Special thanks to Robert L. Thompson, MD FACP, Infectious Disease Section, Group Health Permanente for contributing to this article.

Update: Laboratory Testing and Reporting for West Nile Virus

The following update is to review and update laboratory specimen submission and reporting procedures for West Nile virus (WNV) in King County.

An **Arboviral Encephalitis/Meningitis Case Report Form** is available to health care providers in King County to facilitate the complete and efficient reporting of suspected and confirmed WNV cases in King County residents. Copies of the case report form can be obtained by calling (206) 296-4774, or can be found on the Public Health website at: www.metrokc.gov/health/westnile/

The most efficient diagnostic method is detection of IgM antibody to WNV in serum collected within 8-14 days of illness onset, or cerebral spinal fluid (CSF) collected within 8 days of illness onset using the IgM antibody capture enzyme-linked immunosorbent assay (MAC-ELISA). ELISA testing is available for *hospitalized patients* through the Washington State Public Health Laboratories (WA PHL). Commercial laboratory testing is available for diagnosing patients with suspected mild forms of WNV infection.

Testing at WA PHL can be arranged only after reporting and consultation with Public Health – Seattle & King County. Submit at least 1 cc of cerebrospinal fluid (CSF), and/or separated serum (not whole blood) for ELISA testing. If acute sera and/or CSF specimens are negative, submit a convalescent serum 2-4 weeks after the acute specimen. Specimens should be refrigerated and transported under refrigeration. Frozen CSF is acceptable. Specimens should be submitted with a completed WS PHL *Virus and Rickettsial Examinations* form to the Public Health-Seattle & King County Laboratory, 325 9th Ave, Box 359973, Seattle, WA 98104-2499 (206-731-8950).

WNV cannot be distinguished from other causes of meningoencephalitis on clinical grounds. Therefore, testing for other common causes of aseptic meningitis/encephalitis syndromes, including culture and/or PCR testing for enteroviruses and herpes viruses, is encouraged.

Specimens will be tested for WNV after pending results from tests to identify other etiologies are available.¹

IgM antibody develops by day 8, and IgG antibody develops within 3 weeks after illness onset. When indicated, convalescent serum specimens should be drawn about 3-4 weeks after acute specimens. **Negative results on any specimen obtained <8 days after onset of illness should be considered inconclusive, and a convalescent serum specimen, obtained at least 2 weeks after the first specimen, should be collected and tested.** Cross-reactions may occur among patients who have had yellow fever or Japanese encephalitis vaccination, or a previous history of arboviral encephalitis or dengue fever.

¹ Olin, et al. Aseptic meningitis epidemic during a West Nile virus avian epizootic. *Emerg Infect Dis*, 2003;9:1082-1088. www.cdc.gov/ncidod/EID/vol9no9/03-0068.htm

Disease Reporting

AIDS/HIV (206) 296-4645
 STDs (206) 731-3954
 TB (206) 731-4579
 All Other Notifiable Communicable Diseases (24 hours a day) (206) 296-4774
 Automated reporting line for conditions not immediately notifiable (206) 296-4782

Hotlines

Communicable Disease (206) 296-4949
 HIV/STD (206) 205-STDS

Online Resources

Public Health Home Page: www.metrokc.gov/health/
EPI-LOG Online:
www.metrokc.gov/health/providers
 Subscribe to the Public Health Communicable Disease listserv (PHSKC INFO-X) at:
http://mailman.u.washington.edu/mailman/listinfo/p_hskc-info-x

Reported Cases of Selected Diseases, Seattle & King County 2003

	Cases Reported in August		Cases Reported Through August	
	2003	2002	2003	2002
Campylobacteriosis	24	26	166	210
Cryptosporidiosis	2	4	30	12
Chlamydial infections	372	335	3,314	2,837
Enterohemorrhagic <i>E. coli</i> (non-O157)	0	0	1	0
<i>E. coli</i> O157: H7	3	8	18	16
Giardiasis	12	17	75	125
Gonorrhea	102	100	426	443
<i>Haemophilus influenzae</i> (cases <6 years of age)	0	0	0	0
Hepatitis A	1	1	18	25
Hepatitis B (acute)	1	3	22	20
Hepatitis B (chronic)	44	47	384	341
Hepatitis C (acute)	1	1	7	9
Hepatitis C (chronic, confirmed/probable)	73	112	645	1096
Hepatitis C (chronic, possible)	18	34	161	292
Herpes, genital (primary)	34	48	426	443
HIV and AIDS (includes only AIDS cases not previously reported as HIV)	22	17	296	419
Measles	0	0	0	0
Meningococcal Disease	0	0	3	15
Mumps	0	0	0	0
Pertussis	25	16	168	80
Rubella	0	0	0	2
Rubella, congenital	0	0	0	0
Salmonellosis	19	23	153	141
Shigellosis	4	9	74	40
Syphilis	7	8	57	29
Syphilis, congenital	0	0	0	0
Syphilis, late	3	1	30	24
Tuberculosis	14	12	107	97

The *Epi-Log* is available in alternate formats upon request.