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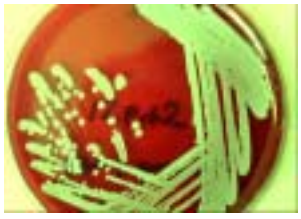
Methicillin-Resistant *Staphylococcus aureus* Skin Infections

Introduction

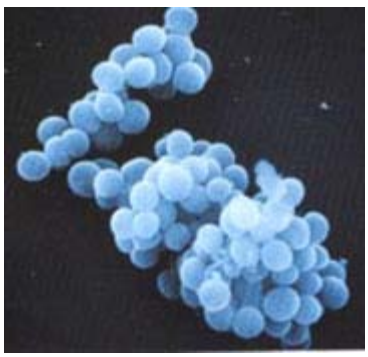


Methicillin-resistant *Staphylococcus aureus* (MRSA) are resistant to the entire class of beta-lactam antibiotics (including penicillin, cloxacillin, dicloxacillin, oxacillin and nafcillin) as well as cephalosporins.[1,2,3] First reported in the 1960s, the prevalence of MRSA has increased since the 1980s, accounting for 30% or more of all *S. aureus* infections in some facilities.[4]

Until recently, MRSA had been uncommon in communities and occurred mostly among persons in hospitals and healthcare facilities. Researchers believed that the antimicrobial selection pressure in healthcare facilities that favored the survival of MRSA actually reduced the organism's ability to compete in the community setting.[5,6]

In fact, MRSA has been emerging in the community over the last several years.[6] While community-associated MRSA (CA-MRSA) usually causes mild and superficial skin infections that respond to proper skin care and antibiotics, aggressive infections (e.g., necrotizing pneumonia) are being observed more frequently, particularly in isolates that carry additional virulence factors.[1,5]

The January 2004 issue of the *Virginia Epidemiology Bulletin* (Vol.104, Issue 1) provided an update on MRSA and special populations in Virginia. Recent anecdotal experience suggests, however, that the frequency of MRSA infections is increasing among otherwise healthy individuals without typical MRSA risk factors.[3] Other developments, such as an early 2005 study that described 14 cases of necrotizing fasciitis due to CA-MRSA in Los Angeles from 2003-2004, have also raised public concern over MRSA.[7] This article is intended to assist clinicians in Virginia in the management of skin and soft tissue infections (SSTIs) until more definitive guidelines are available from other medical professional organizations. The content has been adapted from the Washington State Health Department's interim clinical guidance for the management of *S. aureus* SSTIs in outpatients.[3]



Epidemiology

S. aureus infections include skin infections (e.g., boils, abscesses), osteomyelitis, septic arthritis, endocarditis, meningitis, and pneumonia.[4, 8] Like all *S. aureus*, MRSA can spread among people who have close contact with infected or colonized individuals, usually through direct physical contact. Spread of the organism has also been shown to occur through indirect contact with contaminated objects (e.g., towels, sheets, wound dressings, clothes, workout areas, or sports equipment).[1]

Since individual MRSA infections are not notifiable conditions in Virginia, limited data are available on the incidence and prevalence of colonization or infection. However, studies have suggested that MRSA and CA-MRSA are a significant concern nationwide. For example, at one Los Angeles medical center, 62 percent of community-associated *S. aureus* infections were due to MRSA.[7] And the Washington State Antibiotic Resistance Sentinel Network found an increase from 19% to 35% over two years in the percentage of MRSA among *S. aureus* isolates from outpatients.[9] In addition, some of the recently recognized CA-MRSA outbreaks have been caused by strains with unique properties compared to the traditional healthcare-associated MRSA (HA-MRSA) strains.[1]

Increased risk of developing a MRSA infection in the community has been associated with recent antibiotic use, sharing contaminated items, recurrent skin diseases, and living in crowded settings.[1] Clusters of infection in the U.S. have also occurred among injection drug users, American Indians, incarcerated persons, military recruits, players of close-contact sports, men who have sex

In This Issue:

Factors that increase the level of suspicion for MRSA[3]:

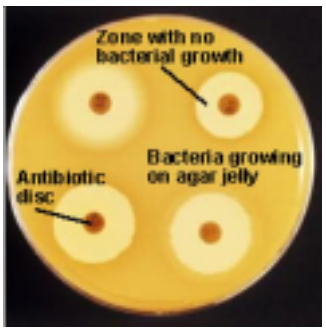
- History of MRSA infection or colonization
- History in the past year of:
  - ➔ Hospitalization
  - ➔ Admission to a long-term care facility (nursing home, skilled nursing, or hospice)
  - ➔ Dialysis and end-stage renal disease
  - ➔ Diabetes mellitus
  - ➔ Surgery
  - ➔ Permanent indwelling catheters or medical devices
  - ➔ Injection drug use
- High prevalence of MRSA in local community or patient population (as indicated by results of local antimicrobial susceptibility testing, clinical experience and surveillance data)
- Recent and/or frequent antibiotic use
- Close contact with someone infected or colonized with MRSA
- Recurrent skin disease (e.g., eczema)
- Incarceration
- Infection among:
  - ➔ Persons living in crowded conditions (e.g., homeless shelters, military recruits)
  - ➔ Sports participants who have:
    - ♦ Skin-to-skin contact
    - ♦ Pre-existing skin damage
    - ♦ Shared clothing and/or equipment
  - ➔ Pacific Islanders, Alaskan Natives, Native Americans
  - ➔ Men who have sex with men
- Poor clinical response to beta-lactam antibiotics

with men (MSM), and other populations.[1,10] Most of the transmission in these settings appeared to be from people with active MRSA skin infections.[1]

Diagnosis

Optimal therapy for SSTIs requires careful evaluation of the patient, including a thorough medical and social history, a physical examination of the lesions, and an examination of Gram-stained smear of exudates or discharges.[2] Clinicians should determine if household or other close contacts of the patient have SSTIs or other infections compatible with MRSA.[3]

Since the bacterial causes of common community-acquired SSTIs are generally Gram-positive organisms such as *S. aureus* and *Streptococcus pyogenes*, most healthcare providers do not routinely obtain cultures of SSTIs. And in general, for mild SSTIs (e.g., furuncles, non-fluctuant boils) where antimicrobial therapy is not anticipated, obtaining cultures may not be necessary for successful treatment. However, routine cultures and antimicrobial sensitivity testing of SSTIs does enable healthcare providers to monitor the extent of CA-MRSA infections in their community and adjust therapy in areas where CA-MRSA is prevalent.[11] Cultures are also advised for acute bacterial skin infections where MRSA is suspected;[12] however, appropriate treatment should not be delayed. If antimicrobials are considered necessary, or may become necessary, culturing lesions may help to guide therapy. A culture of a skin lesion is especially useful in recurrent or persistent cases of skin infection, in cases of antibiotic failure, and in cases that present with advanced or aggressive infections.[1]

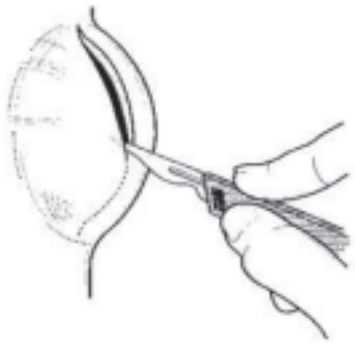


For a frankly pustular lesion, a sterile Dacron swab sample is sufficient; the swab should be placed immediately in broth culture. A small biopsy of skin or aspiration of pus/drainage from the infected site may also be considered.[1] However, unless pus has formed or an open wound is present, the responsible organism may be difficult to isolate. Blood cultures are occasionally positive. If a patient has a history of recurring skin infections, swab specimens of the anterior nares may be considered to check for MRSA colonization.[13]

Of note, if healthcare providers increase their culturing practice, then they are likely to detect more MRSA. This change in practice could lead to the perception that there is a cluster of MRSA occurring in the patient population. Therefore, it is necessary to determine the baseline level of MRSA in a practice's patient population. If an outbreak is suspected (e.g., clustering of cases by time, place or person), the local health department should be contacted. The local health department and the Division of Consolidated Laboratory Services (state laboratory) can help to distinguish unconnected clusters of MRSA from true outbreaks.

Treatment

The clinical approach to treating an SSTI should be based on available information about risk factors for MRSA, the presentation and severity of the infection, and the presence of co-morbidities.[3] **Local treatment and incision and drainage (I & D) remain critical components of therapy for soft tissue infections.** Whether initial therapy with an antibiotic active against MRSA affects the outcome of skin and soft-tissue infection is uncertain. But, with adequate surgical drainage, severe skin and soft-tissue infections often resolve regardless of whether the antimicrobial agent given to the patient has *in vitro* activity.[1,5] As a result, antimicrobial therapy should be reserved for mild infections that cannot be treated with I & D, and for more serious infections (see below).



If antimicrobial therapy is indicated for an SSTI, the initial empiric coverage should be based in part on the prevalence of MRSA in the clinical setting or patient population (ideally guided by local antimicrobial susceptibility patterns for MRSA), as well as the patient's risk factors for MRSA.[2] Therapy should then be modified as needed based on the results of culture and susceptibility testing.[3] Appropriate streamlining of therapy using culture results is important for decreasing morbidity, mortality and the emergence of multi-drug-resistant pathogens.[2]

Management of *S. aureus* SSTIs Based on Severity[3]

- **Mild –** The patient does not have signs or symptoms of systemic toxicity and has no uncontrolled co-morbidities that may complicate treatment (e.g., peripheral vascular disease, diabetes mellitus, chronic venous insufficiency, morbid obesity):
  - o Outpatient management with I & D of abscesses and wound care (with or without topical antimicrobials) and without oral antimicrobial therapy may be an adequate treatment option for many cases.
  - o Antibiotic therapy alone without I & D is not recommended for treatment of fluctuant abscesses.
  - o Consider obtaining specimens for culture and susceptibility testing (especially prior to initiating antimicrobial treatment). If I & D is not performed, other options include culture of spontaneously draining wounds and/or biopsy and culture of the central area of cellulitis. Note: superficial culture of open wounds may yield skin-colonizing bacteria and not the true pathogen.
  - o Consider oral antimicrobials, based on clinical judgment, particularly if I & D is not possible.
    - ♦ If MRSA is not suspected, therapy with a beta-lactam agent (e.g., cephalexin or dicloxacillin) may be adequate. Consider switching treatment within 48 hours if the patient does not improve.
    - ♦ If suspicion for MRSA is high based on the presence of one or more risk factors for MRSA (including a high prevalence of MRSA locally), consider empiric therapy with antimicrobial agents active against MRSA (see Table 1).
  - o Monitor the patient for response to therapy; adjust antimicrobials based on culture and susceptibility results as appropriate.
- **Moderate –** The patient has evidence of systemic illness (e.g., fever) with stable co-morbidities or is systemically well with co-morbidities that may complicate or delay resolution of the infection:
  - o Manage as in- or outpatient, depending on degree of illness and co-morbidity. The patient may require initial hospitalization and parenteral antimicrobials. In areas with a high prevalence of CA-MRSA, or in patients with risk factors for MRSA, empiric treatment with beta-lactam agents may not be appropriate. Switch to oral therapy once signs and symptoms of infection improve.
  - o Monitor outpatients carefully for response to initial oral therapy.
  - o Consider additional cultures (e.g., blood cultures).
  - o Adjust antimicrobials based on culture and susceptibility results.
- **Severe –** The patient appears toxic (e.g., tachycardia, tachypnea, hypotension, altered mental status), or appears non-toxic, but has unstable co-morbidities that could complicate therapy

OR

- **Critically Ill** - The patient has sepsis syndrome or life-threatening infection such as necrotizing fasciitis:
  - o Manage as an inpatient with empiric broad-spectrum parenteral antimicrobial coverage active

Table 1. Interim Guidelines for Empiric Oral Antimicrobial Treatment of Outpatients with Suspected MRSA Skin and Soft Tissue Infections (SSTIs)[3]

Selection of empiric therapy should be guided by patient characteristics and local *S. aureus* susceptibility, and modified based on results of culture with susceptibility testing and clinical response. The duration of therapy for most SSTIs is 7-10 days but may vary depending on severity of infection and clinical response. **NOTE: Before treating, clinicians should consult complete drug prescribing information in the manufacturer's package insert or the Physician's Desk Reference (PDR).**

Antimicrobial	Adult Dose	Pediatric Dose
Trimethoprim-sulfamethoxazole (TMP-SMX) DS	One tablet (160 mg TMP/800 mg SMX) PO BID	8-12 mg TMP/40-60 mg SMX per kg/day in two divided doses; not to exceed adult dose
Minocycline or Doxycycline	100 mg PO BID	*Not recommended for pediatric use - suggest consultation with infectious disease specialist before use*
Clindamycin	300-450 mg PO QID	10-20 mg/kg/day in three or four divided doses; not to exceed adult dose

If considering clindamycin, isolates resistant to erythromycin and sensitive to clindamycin should be evaluated for inducible clindamycin resistance (MLSB phenotype) using the "D test." Consult with your reference laboratory to determine if "D testing" is routine or must be specifically requested. If inducible resistance is present, an alternative agent to clindamycin should be considered, particularly if the clinical response to clindamycin is poor.

against MRSA, including vancomycin.

- Surgical intervention may be necessary.
- Consider additional cultures (e.g., wound, blood, sputum, other).
- Adjust antimicrobials based on culture and susceptibility results.
- Consult an infectious disease specialist if the patient does not improve or alternative antimicrobials (e.g., linezolid) are being considered.
- Discharge to complete a course of outpatient parenteral or oral therapy, based on clinical improvement, tolerance of therapy and availability for follow-up.

Although empiric oral antimicrobial therapy for suspected MRSA infections are shown in Table 1, **there are no data from randomized clinical trials on which to base treatment recommendations**. While CA-MRSA strains are often susceptible *in vitro* to the inexpensive oral agents trimethoprim–sulfamethoxazole (TMP/SMX), doxycycline, and clindamycin, there is limited documented clinical evidence for using these agents to treat MRSA SSTIs.[5] However, because of the high cost and the potential for toxicity and for inducing antimicrobial resistance, linezolid (approved by the Food and Drug Administration for the treatment of MRSA infections) is not recommended for empiric treatment or routine outpatient use.[3,5] Although vancomycin has been the "gold standard" for invasive MRSA infections, most CA-MRSA infections are localized SSTIs that do not require hospitalization or vancomycin therapy.[4] In addition, vancomycin use selects for vancomycin resistant enterococci (VRE), creating the potential for cross-resistance in *S. aureus* since genes conferring vancomycin resistance might be transferred from VRE to *S. aureus*.[4]

Fluoroquinolones (e.g., ciprofloxicin, levofloxacin, moxifloxacin, gatifloxacin) and macrolides (e.g., erythromycin, clarithromycin, azithromycin) are NOT recommended for the treatment of MRSA because of high resistance rates. If fluoroquinolones are being considered, consult with an infectious disease specialist before use.[3]

Additional clinical trials are needed to determine the precise role of antimicrobial agents in the treatment of uncomplicated skin and soft-tissue infections and to identify which agents are most clinically- and cost-effective.[5]

Finally, even if a healthcare provider feels that an antimicrobial is not likely to be necessary, additional factors (e.g., difficult access to health care) may make a prescription a consideration. In these cases, one option is to provide the prescription with instructions to the patient to wait a few more days to see if the infection improves before filling the prescription; ask the patient to destroy the prescription if it is not filled. Always remind patients to complete the entire course of an antimicrobial as directed.

Decolonization

The efficacy of eradicating MRSA colonization (i.e., decolonization) in preventing re-infection or transmission in the outpatient setting has not been documented and is NOT routinely recommended. However, it may be reasonable to consider decolonization for:

- Patients with recurrent MRSA infections despite appropriate therapy; and,
- Ongoing MRSA transmission in a well-defined cohort with close contact (e.g., transmission and infection within a family).

Although the optimal regimens for eradication of colonization have not been established strategies include the following:

- Oral antimicrobials. Possible eradication regimens include rifampin (adult dose: 300 mg PO BID for five days; pediatric dose: 10-20 mg/kg/day in two doses not to exceed 600 mg/d for five days) **in combination with** TMP-SMX **OR** doxycycline **OR** minocycline. Never use rifampin monotherapy, due to the rapid emergence of resistance; and/or,
- Nasal decolonization with intranasal topical mupirocin (BID for five days); and/or,
- Skin antisepsis (e.g., chlorhexidine baths).[3]

NOTE:Rifampin should never be used as a single agent to treat infection or colonization with MRSA

Additional considerations include minimizing the risk of re-colonization through assessing and treating close contacts, intense environmental cleaning, and following prevention strategies (see below). Consultation with an infectious disease specialist before decolonization is attempted is highly recommended.

Prevention

MRSA transmission can be reduced by following these six steps:

1. **Appropriate wound care**[1]: Infections, particularly those that continue to produce pus or to drain, should be kept covered with clean, dry bandages.
2. **Hand hygiene**: Patients, as well as their family and other close contacts, should wash their hands frequently with soap and warm water, especially

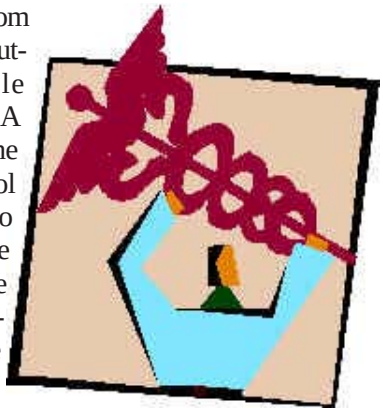
NOTE: Group A streptococci (GAS) are another common cause of SSTIs, particularly cellulitis and impetigo. If GAS infection is suspected, therapy should include an agent active against this organism (ß-lactam, macrolide, clindamycin). Tetracyclines and trimethoprim-sulfamethoxazole, although active against many MRSA, are not recommended treatments for suspected GAS infections.[3]

if contacts change the patient's bandages or touch the infected wound or other potentially infectious materials.[1]

3. **Intense environmental cleaning with appropriate disinfectant cleaners**: Although time and energy intensive, cleaning may be necessary to decrease the levels of MRSA in the environment and reduce the risk of transmission. Environmental sources of MRSA can be extensive: MRSA transmission from animals (e.g., dogs, horses) to humans has been documented.[14]
4. **Avoid sharing personal items**: Items such as towels, washcloths, razors, clothing, or uniforms that may have had contact with the infected wound or potentially infectious material may transmit MRSA. Linens and clothes that become soiled should be washed with hot water and laundry detergent. Drying clothes in a hot dryer, rather than air-drying, also helps kill bacteria in clothes.[1]
5. **Notification of healthcare providers**: Patients should inform their healthcare providers that they may be carriers of MRSA.[1]
6. **Contact precautions**: Patients should avoid contact sports or other skin-to-skin contact until the infection has healed.[3]

Public Health Response

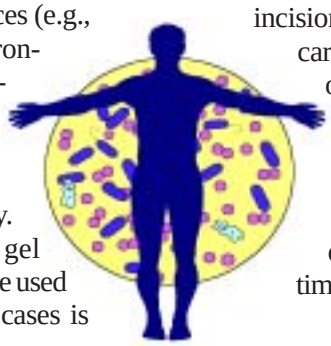
Individual cases of MRSA are not notifiable conditions in Virginia; this affects the ability to monitor changes in the incidence and prevalence of MRSA infection and colonization in Virginia. However, **outbreaks of MRSA infection are reportable to the local health department**. Health departments can assist in the management of MRSA outbreaks by providing advice and resources (e.g., signs and fact sheets, environmental assessments, investigators). Local health departments can also assist in coordinating laboratory testing to determine isolate clonality. For example, pulsed-field gel electrophoresis (PFGE) can be used to determine if a cluster of cases is likely to have originated from a common source (i.e., an outbreak) or from multiple sources. Selected MRSA isolates may be sent to the Centers for Disease Control and Prevention (CDC) to characterize their virulence factors and toxins. The CDC may also provide additional technical assistance to the Virginia Department of Health in the evaluation of unusual cases or clusters of MRSA infections.



In general, healthcare facilities have controlled MRSA outbreaks by promoting hand hygiene, increasing the use of barrier precautions (gowns, gloves), staff education, and patient screening and isolation. Nevertheless, controlling an outbreak of MRSA in a facility can require months of intense effort, use substantial resources (e.g., through increased use of gowns, increased environmental cleaning, increased laboratory support, etc.), impair staff morale, and jeopardize the facility's reputation with the community. Therefore, the prevention of outbreaks through appropriate hand-hygiene programs and infection control is critical.

Summary

Today's environment demands a high index of suspicion for MRSA when managing skin and soft tissue infections. In addition, since 'traditional' risk factors for MRSA may be absent in patients, wound cultures and antimicrobial susceptibility testing are playing a greater role in the management of SSTIs. Culturing also helps to document MRSA levels in the provider's community[5] and to better direct therapy. However, the increasing prevalence of MRSA does not necessarily require more aggressive antimicrobial use. Instead, basic management through incision and drainage and wound care remain the cornerstones of therapy. Appropriate, targeted antimicrobial use will help to reduce the development of resistance and preserve the effectiveness of current (and future) antimicrobials agents.



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MRSA and Brown Recluse Spiders Bites

Misdiagnosis of *Staphylococcus aureus* skin infections as spider bites occurs.[1] This can also lead to the misperception that spiders transmit methicillin-resistant *S. aureus* (MRSA). Furthermore, the misdiagnosis of a skin infection as a spider bite can impede proper treatment[1] and infection control efforts.

In particular, bites (envenomation) by the brown recluse spider (*Loxosceles reclusa*) receive disproportionate attention since they are known by the general public to produce dermonecrotic wounds. Frequently, however, no spider is observed actually inflicting the bite. Other times, a common brown-colored spider is collected from the general vicinity of the proposed bite incident and misidentified.

The limited distribution of *Loxosceles* in the U.S. (see map), the inconsequential resolution of most brown recluse spider bites, and the reticent nature of the spider, suggest that healthcare providers in nonendemic brown recluse regions (e.g., Virginia) should be cautious in implicating brown recluses in idiopathic necrotic wounds. Culturing specimens taken from wounds attributed to spider bites might determine the actual causative agent. These can include arthropod, bacterial (e.g., *S. aureus*), viral, or fungal agents or may be a result of an underlying disease state.[2]

Brown Recluse Spider
(*Loxosceles reclusa*)
Distinguishing characteristics: three pairs of eyes in a semicircle on the forepart of the head; violin-shaped, dark marking behind the eyes with the neck of the violin pointing toward abdomen.

Additional information on the brown recluse spider is available at http://spiders.ucr.edu/, or visit the Spider Myth Site at www.washington.edu/burkemuseum/spidermyth.in dex.html.[1]

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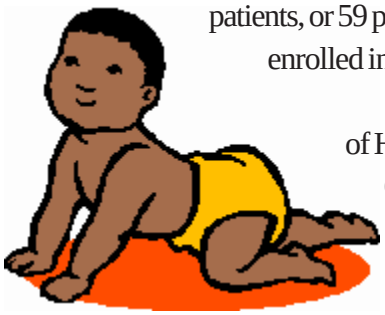
Recluse Spider Populations

Endemic distributions of the brown recluse (stippled) and related recluse species (lines) in the United States. Recluse populations become sporadic on either side of the demarcating range borders.[2]

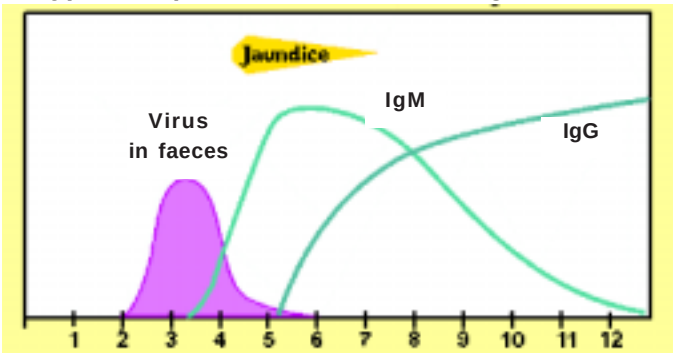
VDH Implements Folic Acid Distribution Program

This July, 19 local health districts will begin to distribute folic acid supplements to family planning and walk-in pregnancy test clinic patients. The goal is to increase the number of women of child-bearing age that take 400 micrograms of folic acid daily. Distribution of the supplement will reinforce the necessity of taking folic acid prior to conception and during the first trimester of pregnancy. Studies have shown that adequate amounts of folic acid can help reduce the risk of spina bifida and anencephaly, the two most common neural tube birth defects, by as much as 70 percent. Approximately 43,000 patients, or 59 percent of the health department's family planning patients, will be enrolled in the program over a 12-month period.



The program was developed jointly by the Virginia Department of Health's Divisions of Child and Adolescent Health, WIC and Community Nutrition Services, and Women's and Infants' Health.

Reducing False-Positive Hepatitis A Testing Results

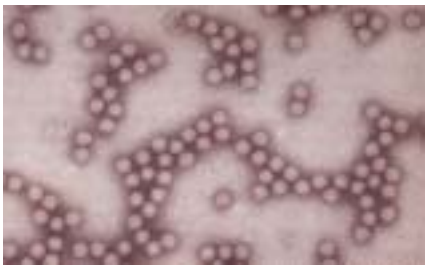


http://web.uct.ac.za/depts/mmi/jmoodie/hepa.gif

Hepatitis A virus (HAV) infection can manifest a broad clinical spectrum, ranging from asymptomatic disease to typical hepatitis with fever and jaundice. Hepatitis A is a reportable condition in Virginia with a surveillance case definition that includes both clinical criteria and serologic confirmation.* However, health departments in Virginia are occasionally notified by laboratories of persons who have positive serologic tests for acute hepatitis A virus (HAV) infection (i.e., IgM anti-HAV), but who do not have illness consistent with the clinical criteria. This situation may occur when laboratory test panels that include IgM anti-HAV are used to evaluate liver function test abnormalities. However, it should be noted that published guidelines for the workup of abnormal liver enzyme tests among asymptomatic patients do not include IgM anti-HAV testing. This article addresses the appropriate use of laboratory testing for HAV to assist healthcare providers in reducing the risk of false-positive tests.

Diagnostic tests for viral hepatitis, including licensed IgM anti-HAV tests, are highly sensitive and specific when used on specimens from persons with acute hepatitis. However, testing of persons without clinical symptoms of acute viral hepatitis and among populations with a low prevalence of acute HAV infection reduces the predictive value of the IgM anti-HAV test. While a positive IgM anti-HAV test result in a person without typical symptoms of hepatitis A might indicate asymptomatic acute HAV infection, it may also represent previous HAV infection with prolonged presence of IgM anti-HAV or a false-positive test result.

In May, 2005, the Centers for Disease Control and Prevention (CDC) published a report in the Morbidity and Mortality Weekly Report (MMWR) on three investigations that resulted from positive tests that may not have represented recent acute HAV infections. Findings in the report indicated that **persons who are unlikely to have acute viral hepatitis should not be tested for IgM anti-HAV.** Further, the report suggests that **the use of IgM anti-HAV as a screening tool or as part of testing panels used in the workup of nonacute liver function abnormalities should be discouraged**. As a result, to improve the predictive value of a positive IgM anti-HAV test, clinicians should limit laboratory testing for acute HAV infection to persons with clinical findings typical of hepatitis A or to persons who have been exposed to settings where HAV transmission is suspected.



If patients without signs or symptoms of hepatitis test positive for IgM anti-HAV, additional testing for total anti-HAV may be helpful to confirm the findings. Persons with acute HAV infection will test total anti-HAV positive. However, if the total anti-HAV test is negative, acute HAV infection is unlikely. Retesting the same or another serum specimen for IgM anti-HAV, preferably by using a different test format, might also help to confirm the original IgM anti-HAV test result.

In summary, healthcare providers should limit use of IgM anti-HAV testing to persons with evidence of clinical hepatitis or to those who have had recent exposure to an HAV-infected person. If hepatitis A infection is suspected, pertinent available clinical information (including date of onset, signs, symptoms, and supporting laboratory data) should be included when the patient is reported to the local health department to enable case classification and appropriate follow-up. Finally, reporting should be by rapid means (e.g., telephone, fax), since if indicated, contacts must receive immune globulin within 14 days of last contact with the infectious case of hepatitis A.

**Acute illness with discrete onset of symptoms (e.g., fatigue, abdominal pain, loss of appetite, intermittent nausea, and vomiting) and jaundice or elevated serum aminotransferase levels. Confirmation requires serologic testing that demonstrates the presence of IgM antibody to hepatitis A virus (anti-HAV), or by identifying recent exposure to a confirmed hepatitis A case.*

Adapted from: Positive Test Results for Acute Hepatitis A Virus Infection Among Persons With No Recent History of Acute Hepatitis—United States, 2002-2004. MMWR. May 13, 2005 / 54(18);453-456

Mark Your Calendar!

Association for Professionals in Infection Control and Epidemiology - Virginia (APIC-VA)

31th Annual Educational Conference

Yesterday, Today and Tomorrow: Crossroads to Change

October 5-6, 2005

Virginia Crossings Resort, Glen Allen, Virginia

Topics will include:

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bonita.allen@hcahealthcare.com

Cases of Selected Notifiable Diseases Reported in Virginia*

Disease	Total Cases Reported, June 2005: State	Regions: NW	N	SW	C	E	Total Cases Reported Statewide, January - June: This Year	Last Year	5 Yr Avg
AIDS	47	4	20	2	9	12	302	359	381
Campylobacteriosis	72	7	21	14	11	19	222	230	236
E. coli O157:H7	7	1	2	2	1	1	14	6	16
Giardiasis	28	5	9	4	8	2	239	170	156
Gonorrhea	727	52	53	112	183	327	4,058	4,257	4,642
Hepatitis, Viral									
A	6	1	3	0	0	2	45	42	54
B, acute	5	1	0	2	2	0	87	99	86
C, acute	2	1	0	1	0	0	9	8	2
HIV Infection	67	8	17	5	18	19	378	427	420
Lead in Children†	63	9	3	14	18	19	229	297	281
Legionellosis	6	2	0	1	0	3	16	9	8
Lyme Disease	17	1	12	0	2	2	50	24	32
Measles	0	0	0	0	0	0	0	0	0
Meningococcal Infection	3	1	0	0	0	2	17	9	21
Mumps	0	0	0	0	0	0	0	3	3
Pertussis	51	26	12	5	3	5	125	73	50
Rabies in Animals	41	8	13	13	5	2	237	233	264
Rocky Mountain Spotted Fever	4	1	0	2	0	1	10	2	3
Rubella	0	0	0	0	0	0	0	0	0
Salmonellosis	73	7	20	19	17	10	372	321	376
Shigellosis	11	0	5	0	5	1	47	49	189
Syphilis, Early§	30	2	8	2	3	15	128	104	113
Tuberculosis	30	3	11	3	10	3	138	98	118

Localities Reporting Animal Rabies This Month: Alexandria 1 bat; Bedford 1 fox; Botetourt 1 skunk; Buckingham 1 cat; Carroll 1raccoon, 1 skunk; Chesapeake 1 raccoon; Chesterfield 1 raccoon; Culpeper 3 raccoons; Fairfax 1 bat, 2 foxes, 3 raccoons; Fauquier 1 bat, 1 raccoon; Frederick 1 raccoon; Grayson 1 raccoon; Henry 1 fox; Loudoun 3 raccoons; Lunenburg 1 skunk; Montgomery 1 raccoon; Nottoway 1 bat; Petersburg 1 raccoon; Pittsylvania 1 raccoon; Prince William 1 fox, 3 raccoons; Roanoke 1 cat; Shenandoah 1 raccoon; Smythe 1 raccoon; Tazewell 1 cat, 1 raccoon; Virginia Beach 1 fox; Warren 1 raccoon; Westmoreland 1 skunk; Wythe 1 cat.

Toxic Substance-related Illnesses: Adult Lead Exposure 5; Pneumoconiosis 7; Silicosis 1.

*Data for 2005 are provisional. †Elevated blood lead levels ≥10µg/dL. §Includes primary, secondary, and early latent.

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