ACINETOBACTER

Revised 02/03/2008

Bacteriology

Acinetobacter are encapsulated, aerobic, non-fermentative gram-negative bacilli. They have a tendency to retain crystal violet that can lead to incorrect identification as gram-positive cocci. Versatility in exploiting a variety of carbon and energy sources allows Acinetobacter to grow on routine laboratory media and accounts for its prevalence in nature.

The lack of characteristics (no color, nonmotile, unable to reduce nitrates, oxidase negative and nonfermenting) has led Acinetobacter to be constantly reclassified. First described in 1908 as Diplococcus mucosus, it has successively been named Micrococcus (small), Mima (mimics), Achromobacter (colorless), Acinetobacter (motionless), and anitratius (nitrate nonreducing).

There are over 20 species of Acinetobacter, though the species Acinetobacter baumannii accounts for more than 80% of isolates causing human disease. Previously all were named Acinetobacter calcoaceticus. They are now divided into 7 species: A. baumannii, A. calcoaceticus, A. haemolyticus, A. johnsonii, A. junii, A. lwoffi and A. radioresistans.

The capsule that surrounds most strains may inhibit phagocytosis and has been speculated to predispose persons with selective complement component deficiencies to infection.

Epidemiology

Reservoir: Acinetobacter is found in almost all soils and water. Acinetobacter has been isolated in a wide variety of foods (pasteurized milk, frozen foods, chilled poultry). Enteral feedings and oral solutions can be contaminated. It can survive for weeks to months on clothing, dry surfaces as bedrails, ventilators and wet surfaces as in sinks. In some patient rooms Acinetobacter is widely spread throughout surfaces that have been in contact with patients, hence the importance of thorough cleaning and disinfection after discharge of an infected patient.

Transmission: Acinetobacter can be spread from person to person (infected or colonized patients), contact with contaminated surfaces of exposure to the environment.

Acinetobacter is a healthcare-related pathogen. Acinetobacter baumannii is primarily a healthcare-associated pathogen. It is increasingly reported as the cause of outbreaks and nosocomial infections such as blood-stream infections, ventilator-associated pneumonia, urinary tract infections and wound infections.

Colonization may occur. It is carried on wet skin areas and nasal cavities but also on dry skin, particularly of health care workers. It is the most common gram-negative organism persistently carried on the skin of
hospital personnel. Up to 25% of healthy ambulatory adults exhibit cutaneous colonization and 7% of adults and infants have transient pharyngeal colonization.

It is also a common colonizer of tracheostomy sites and open wounds. Risk factors for colonization or infection with Acinetobacter include length of hospital stay, surgery, wounds, treatment with broad-spectrum antibiotics, parenteral nutrition, indwelling catheters, mechanical ventilation and admission to an intensive care unit.

Risk factors associated with:
- community-acquired Acinetobacter infection include alcoholism, cigarette smoking, chronic lung disease, diabetes mellitus
- specific for nosocomial infection include length of hospital stay, surgery, wounds, previous infection (independent of previous antibiotic use), fecal colonization with Acinetobacter, treatment with broad-spectrum antibiotics, indwelling central intravenous or urinary catheters, admission to a burn unit or intensive care unit (ICU), parenteral nutrition and mechanical ventilation.

Health care workers are not at risk of disease. Acinetobacter rarely causes serious infection in otherwise healthy people and therefore poses minimal threat to healthcare workers or patients’ family members. Pregnant healthcare workers are not at increased risk from this organism and can therefore care for patients infected or colonized with the organism.

Outbreaks are frequently located in intensive care units and burn units involving patients on mechanical ventilation. Sources of transmission identified in the outbreak setting include predominately respiratory equipment such as resuscitator bags, valves, ventilator circuits, spirometers, peak flow meters, suction catheters, etc. Other sources include humidifiers, warming baths, multidose vials, distilled water, pillows, mattresses, bedpans, showers and water faucet aerators. No source was identified in approximately 50% of reported outbreaks. The costs associated with control of an outbreak can be staggering and some institutions have been forced to close entire units in order to interrupt transmission of Acinetobacter.

Clinical Description

Acinetobacter does not cause disease among healthy individuals. Risk factors for disease include: chronic lung disease, diabetes, weakened immune systems, severe illness. The most common infections are urinary tract, wound and blood stream infections. It can cause suppurative infections in virtually every organ system - meningitis, peritonitis, endocarditis and soft tissue infections. It is the cause of 1% of all nosocomial blood-stream infections and 3% of nosocomial pneumonia in sentinel U.S. hospitals.

The respiratory system is the most common site for Acinetobacter infection because of its transient pharyngeal colonization of healthy persons and a high rate of tracheostomy colonization. Acinetobacter has been reported to cause community-acquired bronchiolitis and tracheobronchitis in healthy children. Tracheobronchitis can also occur in compromised adults. Adult community-acquired Acinetobacter pneumonia generally occurs in patients with diminished host defenses (e.g., alcoholism, tobacco use, diabetes mellitus, renal failure, underlying pulmonary disease).

Acinetobacter is a causative agent of nosocomial pneumonia, particularly ventilator-associated cases. Predisposing factors for nosocomial Acinetobacter pneumonia include endotracheal intubation, tracheostomy, previous antibiotic therapy, ICU residence, recent surgery and underlying pulmonary disease. Nosocomial spread in the ICU setting has been attributed to ventilator equipment, gloves, colonized nursing and respiratory therapy personnel, contaminated parenteral nutrition solution and computer keyboards among others. Nosocomial Acinetobacter pneumonias are frequently multilobar.

Acinetobacter meningitis is rare. Although it is generally identified following head trauma or neuron-surgical procedures, there are reports of Acinetobacter meningitis occurring in healthy hosts. A petechial
rash has been noted in up to 30% of patients with Acinetobacter meningitis. Acinetobacter may be morphologically confused with *N. meningitidis* on Gram stain of spinal fluid.

Acinetobacter is generally thought to be less virulent than many other Gram-negative bacteria. No known cytotoxins are produced.

**Diagnosis**

Infection or colonization with Acinetobacter is usually diagnosed by clinical culture of blood, sputum, urine, wound, sterile body fluid, etc. Microbiologic cultures can be processed by standard methods on routine media.

Antimicrobial susceptibility can be determined by various means, with the agar-dilution method being the gold-standard.

Definitions for “multidrug-resistance” vary widely in the published literature.

**Treatment**

Isolation of Acinetobacter from colonized patients requires no specific therapy.

Acinetobacter isolates demonstrate increasing resistance to commonly prescribed antimicrobials. Resistance has been tracked to plasmids, transposons and chromosomes. Multidrug-resistant *Acinetobacter baumannii* has been reported worldwide and is now recognized as one of the most difficult healthcare-associated infections to control and treat.

Most *A. baumannii* are now resistant to ampicillin, carbenicillin, cefotaxime and chloramphenicol. Resistance to gentamicin, tobramycin and amikacin is increasing. Fluoroquinolones, ceftazidime, trimethoprim-sulfamethoxazole, doxycycline, polymyxin B, colistin, imipenem and meropenem may retain activity against nosocomial Acinetobacter. Broad therapeutic generalizations may not accurately consider local resistance patterns. Therapy must be individualized with observed antimicrobial resistance principles in mind.

Carbapenems (Imipenem and Meropenem) are the mainstay of treatment for antimicrobial-resistant gram-negative infections, though carbapenem-resistant Acinetobacter is increasingly reported. Resistance to the carbapenem class of antibiotics makes multidrug-resistant Acinetobacter infections difficult, if not impossible, to treat.

Colistin and polymyxin B have been used to treat highly resistant Acinetobacter infections. The choice of appropriate therapy is further complicated by the toxicity of colistin which is mainly renal. Acinetobacter isolates resistant to colistin and polymyxin B have also been reported.

Studies have demonstrated in-vitro susceptibility of multidrug-resistant Acinetobacter to various synergistic combinations of antimicrobials including carbapenems, colistin, rifampin and ampicillin-sulbactam. The clinical utility of these combinations against pan-resistant Acinetobacter remains to be determined.
In Louisiana (Antibiogram) the following is a summary of antibiotic sensitivity:

**Total** = Number of samples  
**Average** = average % sensitivity  
**Low** = Lowest sensitivity observed in a hospital  
**High** = Highest sensitivity observed in a hospital

<table>
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<tr>
<th>Group</th>
<th>Antibiotic</th>
<th>Total</th>
<th>% Average</th>
<th>% Low</th>
<th>% High</th>
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<tr>
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<td>Carbenicillin, ticarcillin</td>
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<td>Monobactam</td>
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<tr>
<td>Penicillin &amp; b-lactam Inhib</td>
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<td>Carbapenem</td>
<td>Imipenem</td>
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<td>94.4</td>
<td>80</td>
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<tr>
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<tr>
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<td>Amikacin</td>
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**Prevention**

Due to the high costs generated by outbreaks, there is great incentive to prevent transmission in the healthcare setting and keep the organism from becoming endemic in an institution.

**Standard precautions** should be followed at all times. As with prevention of any healthcare-associated organism, careful hand hygiene should be performed at all appropriate times—either hand washing at the sink or using an alcohol based hand sanitizer. **Contact precautions** are indicated. They should be maintained for the duration of hospitalization or until negative cultures are obtained.

The utility of **active surveillance cultures** to detect patients who are colonized with multidrug-resistant Acinetobacter remains to be determined. Nasal culture screens of high-risk patients may include:
- Ventilator dependent /tracheostomy patients
- Patients admitted from long term care facilities with endemic Acinetobacter
- Patients with previous history of Acinetobacter infection

**Patient placement:** Ideally patients should be placed in a single room or be cohorted. Cohorting of staff may even be considered. In some instances, unit closures occurred.

Supplies should be kept in the patient’s room and discarded after discharge. Whenever possible equipment should be restricted to the patient’s room and not be used for other patients unless thoroughly cleaned and disinfected. Simple “wipe down” of equipment is insufficient. Equipment needs to be disinfected in Central Sterile Supply.

Disinfection of equipment and all surface areas coming into contact with the patient is essential. Terminal cleaning and disinfection of the room is necessary after patient’s discharge.