

Adenovirus

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In Brief

Adenovirus

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Author Disclosure

Drs Bhumbra, Wroblewski, and Serwint have disclosed no financial relationships relevant to this In Brief. This commentary does not contain a discussion of an unapproved/investigative use of a commercial product/device.

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Adenoviruses, so named because they were discovered initially in adenoidal tissue, are DNA viruses that can cause a multitude of clinically significant human disease syndromes. More than 50 different strains of the virus have been

discovered. Most children become infected with at least one strain within the first 5 years after birth, and infection is more likely in child care and overcrowded conditions. Community outbreaks of adenoviral disease have been recognized worldwide. Infection can be asymptomatic, and reinfection also is possible.

Adenovirus is transmitted from **person to person** through contact with respiratory secretions, through fecal-oral transmission, and via **fomites**. Exposure to contaminated swimming pool **water** and lakes has caused outbreaks. Nosocomial transmission in health-care settings from personnel and improperly cleaned equipment also can occur. Adenoviral infection can take place at any time throughout the year, but outbreaks usually are concentrated in winter, spring, and early summer. Although the virus **can survive on surfaces for days, the incubation period varies from 2 to 14 days for respiratory infections and 3 to 10 days for gastrointestinal disease**.

The respiratory and gastrointestinal systems are affected most commonly. Symptoms of respiratory tract infection in children include those associated with a **nonspecific febrile illness, upper respiratory tract infection, otitis media, pharyngitis, exudative tonsillitis, and pneumonia**. **Pharyngoconjunctival fever is characterized by fever, tonsillitis (sometimes suppurative), follicular conjunctivitis, coryza, and diarrhea**. Cervical and preauricular **lymphadenopathy** is common. The presence of a **generalized rash in association with fever, conjunctivitis, and pharyngitis can be mistaken for Kawasaki disease**. Other clinical manifestations of adenoviral

disease include a **pertussislike** illness and **bronchiolitis** obliterans in young infants. Occasionally, especially in immunocompromised children, severe infection can lead to **meningitis, myocarditis, pericarditis, hemorrhagic cystitis, or hepatitis**. Fulminant infections with multiorgan failure can occur in neonates. Gastroenteritis follows infection with enteric adenovirus serotypes 40 or 41 and less often with serotype 31. More recently, severe and fatal infections due to serotype 14 have been reported in all age groups.

Adenoviral infections are highly contagious, but spread can be reduced by using proper hand hygiene, surface cleaning with bleach, and contact and droplet precautions when treating hospitalized patients known to have adenoviral disease. Pharyngoconjunctival fever prevention requires proper chlorination of swimming pools. Infection is most communicable during the initial illness, but virus shedding can persist long after symptoms have resolved.

Several diagnostic techniques can identify an adenoviral infection. The most readily available tests are direct antigen detection and viral isolation in clinical specimens from various body sites and fluids. A rapid antigen test for adenovirus has a high sensitivity and specificity compared with viral culture. Traditional viral culture detection takes longer, with cytopathic effects commonly noted within 1 week. A modification of viral culture known as the rapid shell vial technique can detect virus growth 1 to 2 days after specimen inoculation. Enteric serotypes 40 and 41 are identified best by antigen detection in stool specimens.

Other identification methods include restriction endonuclease analysis,

polymerase chain reaction, serology, and electron microscopy. These techniques are not universally available, despite both restriction endonuclease analysis and polymerase chain reaction being even more sensitive and specific than antigen detection by immunofluorescence. Suitable clinical specimens include nasopharyngeal aspirate or swab, throat swab or wash, rectal swab, conjunctival swab or scrapings, stool, urine, cerebrospinal fluid, and tissue. Specimens should be obtained as early as possible after the onset of disease to increase the chances of virus detection.

Although adenoviral infections generally are self-limited and require no more than supportive care, more severe disease can occur in immunocompromised patients, and antiviral agents

such as ribavirin and cidofovir have been used with inconsistent results.

Comment: The discovery of the clinical manifestations of adenovirus spans the past century. Although viral strains initially were isolated in 1953, epidemics of keratoconjunctivitis were described in Austria as early as 1889, followed by descriptions of pharyngoconjunctival fever in the 1920s and reports of adenovirus strains that caused enteritis in 1975. With such a wide array of presentations, clinicians need to consider adenoviral infections when evaluating all of these symptoms. Although immunocompetent children may be asymptomatic or have self-limited disease, specific diagnosis of adenovirus can be important in certain

circumstances. Because the signs and symptoms of adenovirus may mimic Kawasaki disease, differentiation between Kawasaki and adenovirus is essential because of the time sensitivity of treatment for Kawasaki disease. Precise diagnosis in immunocompromised patients, especially pediatric transplant patients, can be equally important, as is diagnosis during outbreaks or for hospital "cohorting" procedures. When diagnosis is important, polymerase chain reaction and shell viral culture have enhanced sensitivity over rapid immunofluorescence tests; practitioners need to be aware of the types of tests available in the laboratories they use.

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In Brief

Toxic Plants

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More than 60,000 calls are made annually to poison control centers (PCCs) for cases of suspected plant toxicity. Children younger than age 6 years comprise two thirds of cases, due to their natural curiosity and limited judgment. Most of these exposures are benign; fewer than 10% result in treatment by a health professional. The PCC

is a valuable source of information to assist in management.

Most ingestions of plant material by young children are of small quantity, and symptoms, if present, typically are short-lived and self-limited. Gastrointestinal effects are common and may be a clue to seek other, more subtle signs of poisoning. Plant ingestions in older children and adolescents generally are intentional and of larger quantity, the result of either substance experimentation or attempted self-harm.

Autonomic toxidromes can be seen in many plant poisonings. Deadly nightshade (*Atropa belladonna*) and Jimson weed (*Datura stramonium*) produce atropine, scopolamine, and hyoscyamine, all anticholinergic toxins. Victims can present with classic symptoms of flushing, hyperthermia, blurred vision, dry mouth, and hallucinations. Common

2007 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 25th Annual Report. Bron-

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