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ADENOVIRUS IN CHILDREN: CLINICAL SPECTRUM AND OUTCOMES

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Annotation

Human adenoviruses can infect individuals of any age but are most prevalent in children, particularly infants and young children. Most children experience at least one adenovirus infection by the age of 10. Symptoms often resemble the common cold, including runny nose, fever, cough, and sore throat. More severe lower respiratory tract infections—such as bronchitis, bronchiolitis, and pneumonia—can occur and may be life-threatening. Adenoviruses can also lead to conjunctivitis, gastroenteritis, cystitis, myocarditis, cardiomyopathy, and meningoencephalitis. Recent advances in structural and molecular biology have refined adenovirus taxonomy and deepened our insight into the range of associated clinical diseases. Given the broad and complex spectrum of illnesses linked to adenovirus infection, this review focuses primarily on basic virology, epidemiology, and clinicopathological correlations. Since clinical symptoms and pathological findings are inherently connected, examining these relationships within specific adenovirus-related disease entities provides a clearer understanding of this common pediatric viral infection.

Key words

Human adenoviruses, viral infection, clinicopathological.

Introduction. Human adenoviruses (HAdVs) belong to the *Adenoviridae* family, a name originating from their first discovery in human adenoid tissue in 1953. They are medium-sized (70–100 nm), nonenveloped viruses with an icosahedral protein shell (capsid) that houses a double-stranded linear DNA genome.The viral capsid has a precise architecture:

- The shell is primarily built from 240 **hexon** protein trimers.
- At each of the 12 vertices sits a **penton** base capsomere.
- A slender **fiber** protein projects from each penton base. Each of these structural proteins plays a distinct functional and immunological role:
- The **hexon** carries key antigenic markers, determines the virus's group and type, and is the main target for neutralizing antibodies.

• The **penton base** is associated with cellular toxicity and helps the virus escape from endosomes after entering the cell.

• The **fiber** is responsible for the initial attachment to host cell receptors and determines the virus's type specificity. Infection begins when the virus attaches to a cell surface receptor via its fiber, triggering internalization. Once inside the nucleus, the virus commandeers the cell's machinery to transcribe its genes, replicate its DNA, and assemble new viral particles. This replication cycle culminates in cell lysis, releasing new viruses to infect neighboring cells. The process of viral replication and cell destruction produces distinctive microscopic changes in infected tissues, which will be detailed later. Recent progress in techniques like cryo-electron microscopy and atomic force microscopy, combined with the discovery of new viruses, has greatly enhanced our understanding of adenovirus structure and the variations that exist across different species and genera.

Currently, 104 distinct types of human adenovirus (HAdV) have been identified. These are categorized into seven species, labeled A through G, based on criteria such as the guanine-cytosine content of their DNA and other biochemical properties. Traditionally, types up to Ad51 were defined as serotypes. For newer types, a genotypic definition is primarily used, requiring either a novel genetic sequence or evidence of recombination in the genes that code for the major capsid proteins.

Species D contains the largest number of HAdV types (73), followed by species B (16 types). New adenovirus types continue to be discovered.

Human adenoviruses (HAdVs) employ different cellular receptors for entering host cells. For species A, C, D, E, and F (excluding species B), the initial attachment occurs when the fiber knob domain binds with high affinity to the CAR protein, a receptor found abundantly on many tissue types [10]. In contrast, species B HAdVs and species D Ad37 bind to the CD46 receptor, which is present on most human cells, including immune cells.

Adenoviruses possess characteristics that make them highly effective vectors for vaccines and gene therapy. They have a large, well-defined genome suitable for genetic engineering; they do not integrate into the host genome, reducing the risk of mutagenesis; they can be produced in high quantities and are relatively stable; they infect a broad range of cells; and they provoke strong immune responses. Species C HAdVs, such as the widely studied Ad5, are particularly noted for their ability to establish latent infections in lymphoid tissues and are commonly used as non-replicating vectors, including in recent COVID-19 vaccines.

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While generally safe, adenovirus-based vaccines can have side effects. For instance, Ad5 vectors can transiently bind and activate platelets, potentially leading to temporary thrombocytopenia, though the overall risk remains very low.

Epidemiology. Adenoviruses are common in humans and animals, causing sporadic or epidemic infections. Serological studies show that antibodies to types Ad1, Ad2, and Ad5 are most prevalent, found in 40%–60% of young children. Infection rates peak between 6 months and 5 years of age, with most children developing antibodies to common types by age 5. Neonates initially have maternal antibodies, which wane, leading to new infections as they grow. A comprehensive review of 65 years of global seroprevalence data is summarized by Mennechet et al.

Different HAdV types exhibit distinct tissue preferences and cause varied diseases. Young children under 5 are especially susceptible due to time spent in group settings like daycare centers. HAdVs are responsible for 7%–8% of viral respiratory illnesses in this age group. Beyond the respiratory system, adenoviruses are also associated with diseases in the gastrointestinal tract, eyes, urinary system, and nervous system.

Human adenovirus (HAdV) infection disrupts normal cell function by halting the synthesis of cellular DNA, RNA, and proteins. This leads to distinctive degenerative changes in infected cells, which can be key to pathological diagnosis when tissue samples are available.

The process unfolds as follows:

- Host DNA production ceases 8–10 hours post-infection, followed by a shutdown of host protein synthesis 6–10 hours later.
- Viral assembly is inefficient, leaving a large surplus of unassembled viral components to accumulate in the cell nucleus.
- These excess components form characteristic nuclear "inclusion bodies." Two main types are seen: a large, amorphous basophilic mass in "smudge cells," and a bar-shaped, eosinophilic crystal known as a "Cowdry type A inclusion".
- Ultimately, the infected cell ruptures, releasing new virus particles and causing tissue damage. This results in a necrotizing inflammatory pattern with cell debris, fibrinous exudate, and a mix of inflammatory cells. The host's immune response, involving neutrophils, lymphocytes, macrophages, and natural killer cells, along with cytokine release, contributes to both viral control and the symptoms of infection, though specific mechanisms require further study.

Meningoencephalitis is a rare but serious complication of HAdV infection, primarily occurring in immunocompromised individuals such as those with AIDS or lymphoma. While not common, HAdVs have been directly detected in

cerebrospinal fluid. Neurological symptoms range from mild aseptic meningitis to severe, often fatal, acute necrotizing encephalopathy.

Diagnosis can be challenging. Some cases, particularly those linked to epidemic Ad7 pneumonia in children, are diagnosed based on viral isolation from other sites or rising antibody titers. This leaves it unclear whether neurological symptoms are due to direct brain infection or an indirect effect. In rare fatal cases, however, characteristic "smudge cells" can be found within neurons of the brain, and direct viral infection can be confirmed using a specific anti-adenovirus immunohistochemical (IHC) stain.

Acute follicular conjunctivitis is the most common form of adenoviral ocular infections. The infection is usually confined to one eye and appears as follicular lesions on the palpebral conjunctival surface. Symptoms usually occur after an incubation period of 5–7 days and include itching, burning, lacrimation, discharge, foreign body sensation, and prominent conjunctival congestion. Physical examination shows erythema and lymphoid follicular hyperplasia in the conjunctiva in association with serous drainage and increased lacrimation. Occasionally, adenopathy of the preauricular lymph nodes is seen. Symptoms usually resolve in 10 days to 3 weeks with complete recovery.

Pharyngoconjunctival fever (PCF) is a syndrome characterized by pharyngitis, conjunctivitis, and fever. Not all patients have the complete syndrome triad during epidemics. The usual onset of illness is abrupt, with sore throat, eye irritation or pain, fever, and generalized soreness. Photophobia and lacrimation are unusual. Many patients develop cough from catarrhal inflammation of the nasal mucosa and posterior nasal discharge. On examination, the palpebral conjunctiva usually appears granular, and hemorrhages occasionally are noted on the bulbar surface. The tonsils, adenoids, and pharyngeal lymphoid tissue are often enlarged with various degrees of pharyngeal congestion, which may result in nasal blockage. Approximately one-third of affected patients show follicular exudative lesions that is similar to streptococcal disease on clinical presentations. Involvement frequently starts in one eye and does not involve the other eye until 2 or 3 days later. The fever is sustained or remittent for 3–4 days in most patients. Throat and eye findings usually are improved considerably by the seventh day of illness, but some constitutional symptoms may persist for two weeks or longer.

Epidemic keratoconjunctivitis (EKC) is caused most commonly by Ad8 and Ad37. It occurs more commonly in adults than children. The usual mode of viral spread is by contaminated ophthalmic instruments and eye solutions, hand-to-eye contact by infected personnel, swimming pools, or fomites in close-contact situations. The incubation period typically is 5–10 days. The initial symptom

generally is unilateral follicular conjunctivitis with a foreign body sensation. Photophobia, lacrimation, discharge, hyperemia, and edema of the conjunctiva are notable. Preauricular adenopathy is frequently present and many of those afflicted also have pharyngitis and rhinitis. The conjunctivitis resolves 7–10 days after onset of the disease. In severe cases, blurred vision may continue for a long period of time. An infantile form of EKC has been described that usually affects children younger than 2 years old. High fever, pharyngitis, otitis media, diarrhea, and vomiting usually accompany this form of pseudomembranous or membranous conjunctivitis. In acute illness, conjunctival scrapings obtained during the first 10 days of infection reveal characteristic inclusion bodies when stained with Giemsa or Papanicolaou stain. Intranuclear inclusions with a ground-glass nuclear appearance can be demonstrated. The conjunctival smears usually show inflammation with predominantly lymphocytes and little fibrinous discharge. Virus-specific fluorescent antibody staining is diagnostic in EKC. Preparations of corneal and conjunctival epithelia reveal adenoviral particles when examined with the electron microscope.

Although HAdVs have been frequently implicated as an etiologic agent in common colds, only 3%–6% of common colds in children were attributed to HAdVs. Adenoviral pharyngitis is an acute illness characterized by fever, sore throat, extensive exudative tonsillitis, and frequently, cervical adenopathy. Other associated symptoms include headache, myalgia, chills, malaise, and cough. In infants and preschool children, nasal congestion and discharge are more prominent, and abdominal pain is a common complaint. Acute febrile pharyngitis is the most common adenoviral illness in children and is particularly important as an epidemic illness in closed environments. On occasion, HAdVs have been associated with acute laryngotracheitis. In general, the symptoms caused are not severe and usually only presents with a barking and harsh cough. Laryngotracheitis frequently is seen in association with febrile pharyngitis, bronchiolitis, and pneumonia.

Recently, a 3-year cross-sectional hospital-based study showed HAdV species B, C, and E were detectable in adenoid and palatine tonsil tissues and nasopharyngeal secretions from nearly 85% of children with adenotonsillar hyperplasia or recurrent tonsillitis. There is no association with the severity of airway obstruction, nor with the presence of recurrent tonsillitis, sleep apnea or otitis media. The histopathology shows follicular hyperplasia with increased lymphocytes and macrophages. No characteristic viral inclusions are observed. Epithelial and subepithelial cells in tonsils seem to be crucial for HAdV species C production and shedding in such persistent HAdV infection.

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