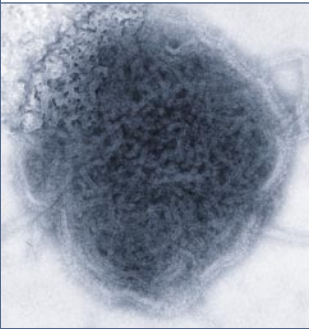


Parainfluenza Virus Overview

ABOUT THE VIRUS

The human parainfluenza viruses (HPIVs) consist of four serotypes, 1-4. They are negative-sense, single-stranded RNA viruses of the Paramyxoviridae family. The HPIVs were first discovered between 1956 and 1960. HPIV1, HPIV2, and HPIV3 were isolated from infants and children with lower respiratory tract disease; HPIV4 was isolated from children and young adults with mild upper respiratory disease. HPIV infection is one of the leading causes of lower respiratory tract disease among children, second only to respiratory syncytial virus (RSV). The four serotypes of HPIV differ in seasonality, prevalence, and clinical disease. HPIV1 and 2 are the leading causes of childhood laryngotracheobronchitis (LTB) and are most prevalent in the fall and winter months. HPIV3 is found year-round, but it is most prevalent in the spring and early summer months. HPIV3 is more often associated with adult disease and bronchiolitis or pneumonia, especially in the elderly and immunocompromised patients. HPIV4 is not frequently detected and has not been well characterized.



Paramyxoviridae virus viewed by transmission electron micrograph.
Courtesy of CDC/Dr. Fred Murphy.

CLINICAL MANIFESTATIONS

HPIV is a common community acquired infection in children and presents as rhinitis, pharyngitis, cough and hoarseness with a fever that lasts approximately 2-3 days. Otitis media occurs frequently and the virus can be detected in the middle ear fluid of these patients. The initial symptoms progress when acute LTB develops, which varies in intensity, but typically lasts 48-72 hours. After a few days, the cough worsens, becoming barking and seal-like. The infection progresses to the lower respiratory tract approximately 15% of the time; fever and productive cough are accompanied by wheeze, retractions, tachypnea and, in severe cases, cyanosis. Chest radiographs show interstitial or perihilar infiltrates and air trapping. Children may develop bronchopneumonia-croup syndrome or severe pulmonary disease following HPIV infection that resembles adult respiratory distress syndrome.

Infections in immunocompetent adults, who have developed incomplete immunity, tend to be mild and restricted to the upper respiratory tract. However, HPIV infections can cause prolonged and significant disease in immunocompromised patients. HPIV3 is the most common serotype isolated in the transplant population and initially presents as an upper respiratory tract infection with cough as the main symptom; rhinorrhoea, wheeze, coryza and fever are less common. The infection may progress to the lower respiratory tract and lead to pneumonia and respiratory failure; this occurs in approximately one-third of hematopoietic stem cell transplant (HSCT) patients. Lung transplant patients are at higher risk for lower respiratory tract infection. Corticosteroid treatment for graft-versus-host disease (GVHD) is a significant risk factor for progression to the lower respiratory tract. Pulmonary copathogens, particularly *A. fumigates*, are commonly isolated from patients with HPIV pneumonia, which significantly contributes to the mortality rate. Bronchiolitis obliterans and allograft rejection in lung transplant recipients are commonly associated with HPIV infections. Rates of infection between autologous and allogeneic HSCT recipients seem to be similar, though there are limited studies on HPIV infection in the transplant community.

LABORATORY DIAGNOSIS

Viral culture is a common method of HPIV detection, though it is time consuming, taking 3-14 days. Fluorescent antibody techniques are more timely, though poor negative predictive values are a significant limitation of this method. In addition, the tendency of adults to shed low titres of the virus adds to the method's limitations. The need for a rapid and highly sensitive diagnostic method is significant. Polymerase Chain Reaction (PCR) is coming to the forefront as it has been shown to be a rapid, sensitive and specific method for detecting HPIV.

TREATMENT

HPIV is mainly a community acquired infection, though nosocomial spread does occur. Spread of infection can be prevented with careful hand hygiene practices, as inoculation occurs through direct contact with fomites or other infected objects. Isolation measures can help prevent spread of infection by close contact and respiratory droplet transmission; however, asymptomatic viral shedding and an incubation period of 1-7 days can make this challenging.

Treatment options for HPIV infection are limited. Clinical studies of ribavirin, with or without IVIG, have yielded mixed results. Bacterial and fungal coinfections should be carefully monitored.

Selected References

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