Other Viral Pneumonias

Coronavirus, Respiratory Syncytial Virus, Adenovirus, Hantavirus

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KEYWORDS

• SARS • Coronavirus • RSV • Adenovirus • Hantavirus • Acute respiratory failure • Immunocompromised host

KEY POINTS

- Severe acute respiratory syndrome–associated coronavirus and the Middle East respiratory syndrome coronavirus are novel pathogens that can cause severe respiratory infections and acute respiratory distress syndrome, which is associated with high mortality.
- Sustained human-to-human transmission of coronavirus can occur; thus, early case recognition, laboratory diagnosis, isolation, and implementation of appropriate infection control measures in the health care setting are important to prevent disease transmission.
- The diagnosis of respiratory syncytial virus in adults can be challenging and there are no vaccines or antivirals available; these unmet needs should be urgently addressed.
- The diagnosis and surveillance of adenovirus infection has been greatly improved by the development of highly sensitive and quantitative polymerase chain reaction assays of mucosal samples or plasma.
- The diagnosis of hantavirus pulmonary syndrome is primarily based on a history of exposure to potentially infected rodents in endemic areas such as the rural southwestern United States and may be confirmed by polymerase chain reaction or serologic testing.
- Clinical management of hantavirus pulmonary syndrome includes excellent supportive care with particular attention to careful management of fluid status.

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HUMAN CORONAVIRUS INFECTIONS

Virology

Most human coronaviruses (eg, hCoV 229E, OC43, NL63) cause mild upper respiratory tract diseases, except occasionally in immunocompromised hosts. However, 2 novel coronaviruses, the severe acute respiratory syndrome–associated coronavirus (SARS-CoV), and a recently identified Middle East respiratory syndrome coronavirus (MERS-CoV) may cause serious viral pneumonitis, leading to hospitalization and death.[1,2] Coronaviruses are large, lipid-enveloped, positive-sense, single-stranded RNA viruses. Viral genome analyses revealed that SARS-CoV and MERS-CoV are Group B and Group C betacoronavirus, respectively, and are closely related to coronaviruses found in bats.[1–4] Intermediate mammalian hosts such as civet cats have been implicated for SARS-CoV before its adaptation for human transmission,[1,3] but no such host has been identified for MERS-CoV. These coronaviruses encode a surface spike glycoprotein (S protein) that attaches the virus to host cells, determining its host range and tropism, and is the target for neutralizing antibodies.[1,3] It has been shown that SARS-CoV uses human angiotensin-converting enzyme 2 (ACE-II) as the primary cellular receptor; the human C-type lectin (DC/L-SIGN) has also been implicated as an alternative receptor.[1,3,5] MERS-CoV has been shown to bind to dipeptidyl peptidase 4 (DPP4; also called CD26), an interspecies-conserved protein found on the surface of several cell types including the nonciliated cells in human airways,[4–6] and this interaction may explain its broad host range and its ability to cause cross-species zoonotic transmission.[4] There is no vaccine available at present for coronaviruses.[7]

Epidemiology and Disease Transmission

SARS-CoV emerged in Southern China (Guangdong Province) in February 2003; the first victims were those who had direct contact with live animals, either in the wet markets or in restaurants selling these animals as winter food.[1,3] The disease quickly spread to Hong Kong; and within a few weeks, through international air travel, it had reached Vietnam, Singapore, Taiwan, and Canada.[1,3,8] By July 2003, more than 30 countries were affected, resulting in 8096 confirmed infections and 774 deaths (9.6%).[8] Mathematical modeling of the early phase of the outbreak estimated that the basic reproductive number (R_0) of SARS-CoV was in the range of 2.2 to 3.7; the primary mode of transmission was via respiratory droplets.[9] Two key epidemiologic features of SARS were frequent nosocomial outbreaks and superspreading events, which exacerbated its transmission. Notably, 1706 of 8096 (21%) of SARS victims were health care workers.[8] It has been suggested that viral replication was at its peak in SARS patients at the time of hospitalization when symptoms worsen (see later discussion). Transmission was facilitated by close bed proximity and the application of aerosol-generating procedures (eg, intubation, resuscitation) and devices (eg, continuous positive airway pressure and biphasic positive airway pressure [BiPAP] treatments).[10] In 1 example, nebulization from a bronchodilator in a SARS patient resulted in a major hospital outbreak involving 138 inpatients, doctors, nurses, allied health workers, and medical students who had worked in the same medical ward.[11] Subsequent studies indicated that the attack rates were between 10% and 60% in the hospital settings.[10] An example of a community superspreading event that involved more than 300 residents occurred in a private housing estate (Amoy Gardens) in Hong Kong. Drying up of the U-shaped bathroom floor drain and backflow of contaminated sewage (from a SARS patient with diarrhea), coupled with the toilet's exhaust fan, might have created infectious aerosols that moved upward through the