REVIEW ARTICLE

Andes Virus — A Clinical Review

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SUMMARY

Andes virus (ANDV) is the sole orthohantavirus with documented human-to-human transmission. We summarize the epidemiology and clinical features of ANDV infection and review best practices in clinical management, as based on published expert consensus guidelines, field experience, and clinical trials. We also evaluate currently available and investigational treatments (including the use of antiviral agents), assess emerging monoclonal antibody therapies, and outline prospects for vaccine development. Finally, we discuss important infection prevention and control measures.

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Andes virus (ANDV) is a member of the species *Orthohantavirus andesense* and the only orthohantavirus with documented human-to-human transmission. Orthohantaviruses are trisegmented, negative-sense, single-stranded RNA viruses.[1] Rodents are the primary natural reservoir, although the virus has also been found in other small mammals, including moles and shrews. Like other orthohantaviruses in the Western hemisphere, infection with ANDV may cause hantavirus pulmonary syndrome (previously also known as hantavirus cardiopulmonary syndrome), which is associated with a high case fatality rate (25 to 40%). Survival depends on host and viral factors; early identification of hantavirus pulmonary syndrome followed by initiation of optimal supportive care has been associated with better outcomes in severe cases.[1-7] This review summarizes the epidemiology, clinical features, diagnostics, infection-prevention considerations, and emerging therapeutics for ANDV infection.

EPIDEMIOLOGY

The first known cases of ANDV infection in humans were documented in Argentina and Chile in 1995, after which hantavirus pulmonary syndrome was recognized as an endemic zoonosis in the Southern Cone of South America.[7-9] Cases of human infection cluster in focal areas where ANDV is hyperendemic, but the majority of cases of hantavirus pulmonary syndrome across Argentina and Chile appear to be caused by other orthohantaviruses.[1,7,10-13] Human infection primarily occurs through inhalation of aerosolized rodent excreta or direct contact with contaminated materials (Fig. 1). Cases have traditionally been associated with exposure to rodent-infested rural dwellings or structures, landfills, dumps, or poorly ventilated spaces where aerosolization of contaminated dust may occur. Less common routes of human infection include inoculation during rodent bites and human-to-human transmission.[3]

Observations of household and nosocomial clusters of ANDV infection have implicated prolonged or extensive contact with a person infected with ANDV as a

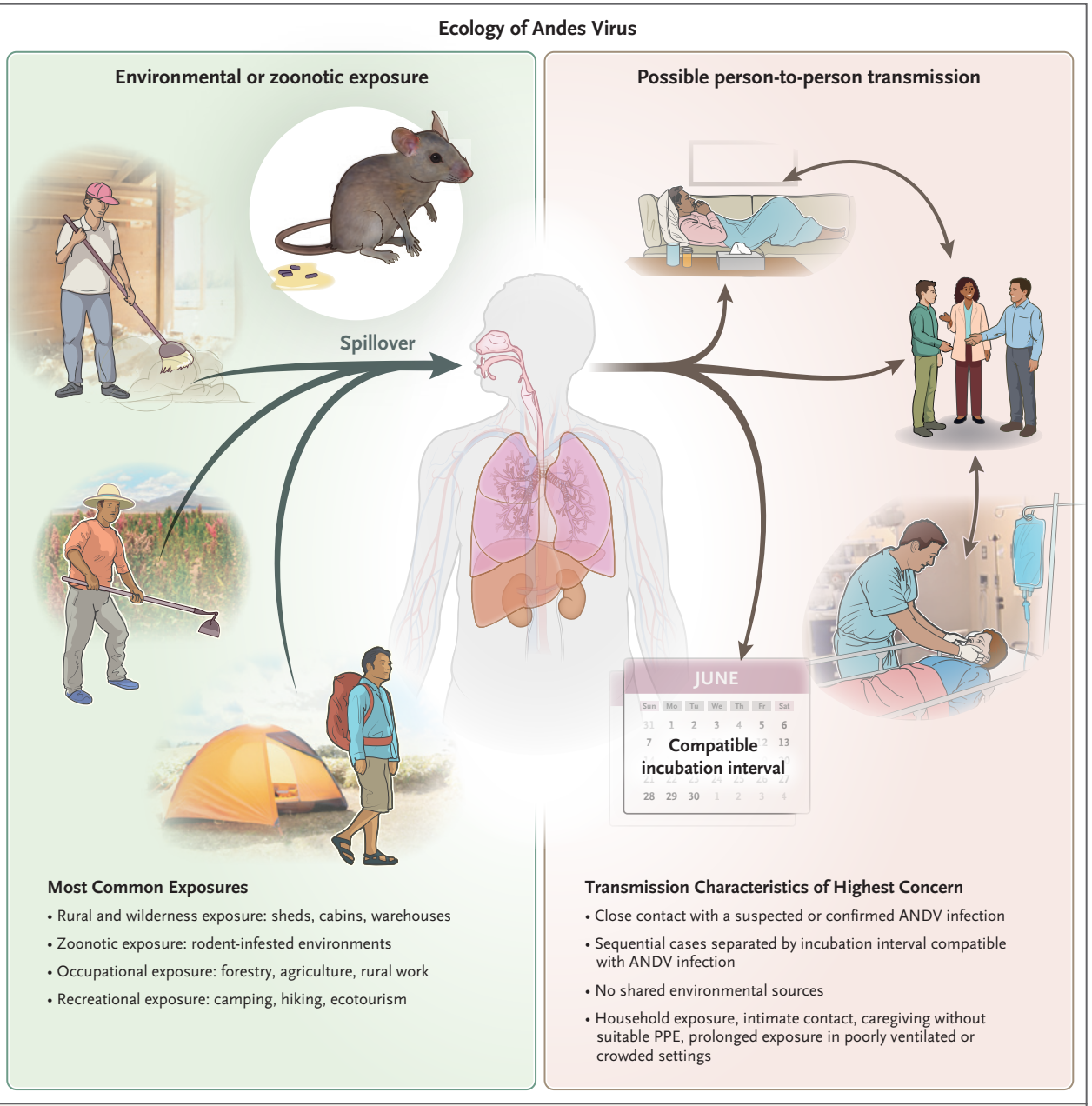

Figure 1. Ecology of Andes Virus.

risk factor for human-to-human transmission.[3] Human-to-human transmission of ANDV has been documented in both Argentina (notably the Epuyén outbreak in 2018 and 2019) and Chile (household and nosocomial clusters). Human transmission has also been linked to the related Buenos Aires genotype (HU39694), but the effect of different variants on transmission is unclear.[3,4] Nevertheless, human-to-human transmission is generally thought to be uncommon and is associated with household exposure, intimate contact, caregiving without suitable personal protective equipment (PPE), and prolonged exposure in poorly ventilated or crowded settings. No evidence supporting efficient community transmission of ANDV exists.[3-7,12]

Ferres et al. monitored 476 contacts of 76 index patients with confirmed ANDV infection in Chile for 5 weeks and observed a secondary infection rate of approximately 3%, with the high-

est identified risk being sexual partnership.[15] Of the 16 patients in their report who had secondary ANDV infection, 6 were reported to have had detectable viral RNA in peripheral blood before symptom onset; viral RNA was detected in some patients for up to 2 weeks before the appearance of symptoms. In a separate report by Ferres et al., samples obtained from blood and gingival crevices after symptom onset tested positive for ANDV RNA in most patients.[5] Saliva, nasopharyngeal, and urine specimens were positive in a minority of patients, with some testing positive up to 3 weeks after symptom onset.

Perhaps the most well-analyzed cluster of human cases occurred in Epuyén, Argentina, during 2018 and 2019. Investigation of that cluster with the use of molecular-genetics techniques revealed three separate superspreader events involving human-to-human transmission, some of which occurred in social settings with casual contact over a short duration. In addition, the risk of transmission appeared to be greatest during the first 1 to 2 days of symptoms in the index patients. No infection in health care workers was documented during that outbreak, despite 82 exposures and inconsistent adherence to recommended infection prevention and control practices.[3]

PATHOGENESIS

After inhalation, ANDV infects the respiratory epithelium and endothelial cells.[9,12,14] Increased pulmonary vascular permeability due to endothelial dysfunction is the hallmark of hantavirus pulmonary syndrome and is associated with high viral loads and dysregulated inflammatory mediators, such as tumor necrosis factor, interleukin-6, and interferon-γ.[2,9,12] Pathological studies show minimal pulmonary cell necrosis despite marked alveolar and interstitial edema and hemoconcentration with considerable inflammatory-cell infiltration.[2,9,12] Once the integrity of the endothelial barrier is lost, intravascular volume diminishes, a factor that is an important contributor to hypotension, shock, and hypoxemia.

CLINICAL FEATURES

Rapidly progressive or severe respiratory compromise in a previously healthy person with a relevant epidemiologic exposure should raise clinical suspicion for ANDV infection. The clinical course of hantavirus pulmonary syndrome — the most severe form of disease due to ANDV infection — is shown in Figure 2. Hantavirus pulmonary syndrome has several phases and begins with a potentially long incubation period (range, 1 to 6 weeks; median, 2 to 3 weeks).[15-19] Symptoms generally begin with a febrile prodrome that can last 1 to 7 days. The prodrome is often associated with headache and severe myalgias and lacks prominent respiratory features. As the disease progresses, frank acute respiratory distress syndrome develops along with circulatory shock; cardiorespiratory collapse and death can occur within 24 to 48 hours without aggressive supportive care optimized for hantavirus pulmonary syndrome. The case fatality rate varies between 20 and 40%.[15-20] Rapidly progressive thrombocytopenia and increased immunoblast percentage (>10%) are early laboratory markers suggestive of hantavirus pulmonary syndrome. Poor prognostic signs for patients needing intensive care include a high lactate level (>4.0 mmol per liter), a low cardiac index (<2.2 liters per minute per square meter of body-surface area), and a worsening hemoconcentration. Proteinuria has also been suggested as a prognostic factor for worse outcomes.[20] Patients surviving to the convalescent phase often show brisk improvement with spontaneous diuresis.[16,17] Complete recovery among survivors occurs 2 to 3 weeks after resolution of the cardiopulmonary phase of infection.[16-19,21]

DIAGNOSTIC TESTING

During the prodromal phase of disease, ANDV should be suspected in patients with thrombocytopenia, marked leukocytosis, and circulating immunoblasts, alone or in combination, and an exposure compatible with the epidemiologic features of ANDV transmission. Hemoconcentration is a common finding in the later stages of disease.[16] Clinicians should notify public health officials about suspected cases to arrange for orthohantavirus testing. Early symptoms of ANDV infection overlap with those of many tropical and seasonal illnesses, and differential diagnoses should include influenza, betacoronavirus infection, legionella infection, mycoplasma infection, Q fever, leptospirosis, severe malaria, rickettsioses, and dengue.

A definitive diagnosis of ANDV infection relies on molecular and serologic testing, which is typically conducted at designated reference laboratories. Because viremia is highest during the

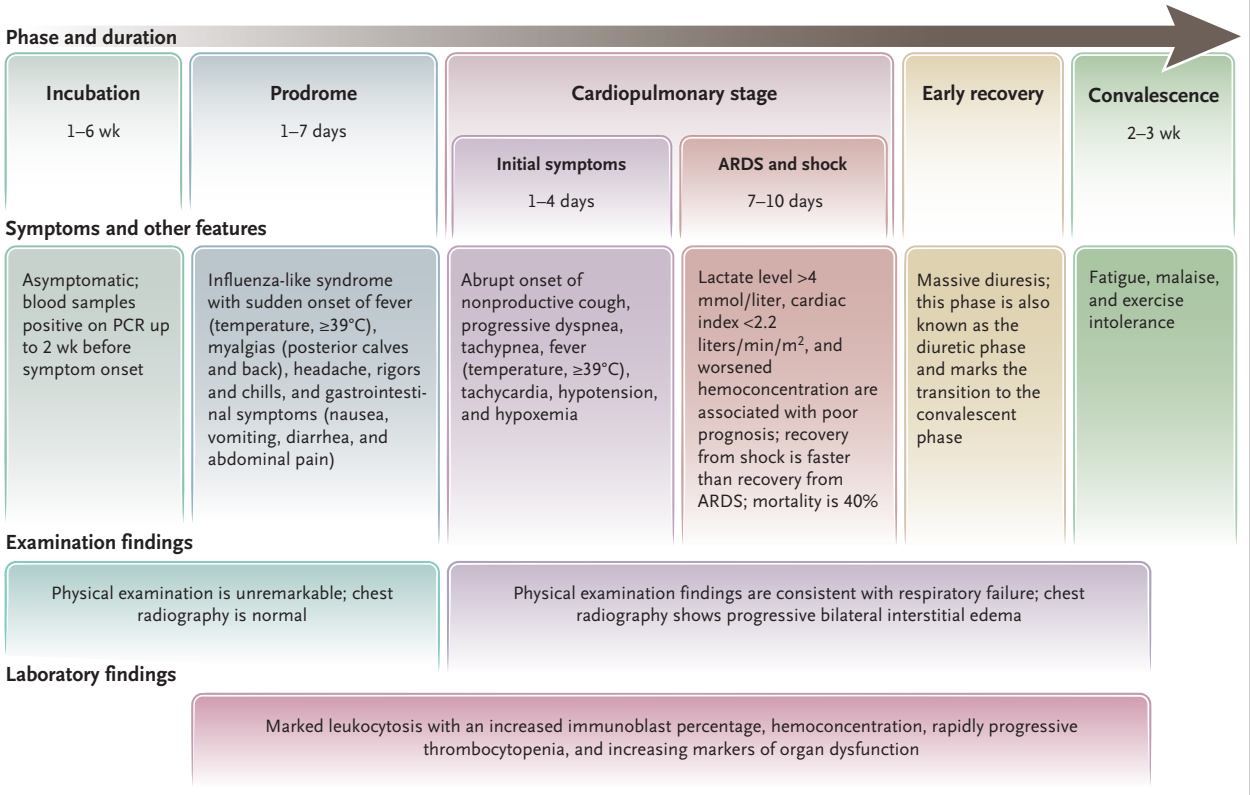

Figure 2. Clinical Course of Hantavirus Pulmonary Syndrome.

During the cardiopulmonary phase, acute respiratory distress syndrome (ARDS) and shock occur in patients with the most severe progressive form of hantavirus pulmonary syndrome, potentially resulting in the use of venoarterial extracorporeal membrane oxygenation in selected patients. PCR denotes polymerase chain reaction.

latter part of the prodromal phase and the early part of the cardiopulmonary phase, reverse-transcriptase–polymerase-chain-reaction assay can detect ANDV RNA in whole-blood samples as early as 2 weeks before symptom onset and is most sensitive in the first week of illness.[15-19,21] Enzyme-linked immunosorbent assays and immunofluorescence assays for ANDV-specific IgM and IgG (against the nucleocapsid protein) have high sensitivity and specificity and are mainstay diagnostic tools, especially in regions where ANDV is endemic. IgM typically appears during the first week of illness and may persist for weeks.[15] Surveillance criteria from the Centers for Disease Control and Prevention (CDC) are summarized in Table 1. A definitive diagnosis of ANDV infection must also consider the possibility of exposure to a person with suspected or confirmed ANDV infection.

MANAGEMENT

No licensed vaccines or targeted therapeutics for ANDV-associated hantavirus pulmonary syndrome currently exist, and no specific drug or immunomodulatory therapy has sufficient evidence to support routine use. The published evidence on specific therapies for ANDV infection consists of two randomized, controlled trials — one that assessed ribavirin and one that assessed methylprednisolone — with negative findings, one nonrandomized open study of convalescent plasma, and several small case reports and case series of newer agents. Thus, because hantavirus pulmonary syndrome can rapidly progress within hours to days, early recognition and prompt supportive care in an intensive care unit (ICU) are essential for survival. In fact, the only intervention with a clear positive recommendation is advanced sup-

portive care, which includes judicious and often restrictive fluid-management approaches, accurate cardiovascular monitoring and assessment, and use of vasoactive agents. Judicious fluid management is recommended because overly aggressive replacement may exacerbate pulmonary edema.[16,17] In selected patients, early venoarterial extracorporeal membrane oxygenation (ECMO) has been associated with high survival in case series.[20,21,23-26] Evidence for venovenous ECMO is lacking.[16,20,25-27] Intubation may precipitate hemodynamic collapse due to profound diastolic dysfunction and suppressed myocardial function.[16,17]

On the basis of a randomized, controlled trial of methylprednisolone and anecdotal reports of the management of non-ANDV hantavirus infections, glucocorticoids have not been recommended.[16,28,29] In a Chilean trial of high-dose intravenous methylprednisolone in patients with ANDV-associated hantavirus pulmonary syndrome, glucocorticoids did not result in a significant clinical benefit or a difference in mortality as compared with placebo (95% confidence interval, 0.32 to 1.39).[30] Guidance from 2007 explicitly lists glucocorticoids among contraindicated agents in patients with exposure to hantavirus.[30,31]

Table 1. Criteria for Surveillance of Hantavirus Pulmonary Syndrome.*

Clinical criteria
Acute febrile illness with a prodrome consisting of fever, chills, myalgias, headaches, and gastrointestinal symptoms and one or more of the following:
Bilateral diffuse interstitial pulmonary edema
Clinical diagnosis of acute respiratory distress syndrome
Radiographic evidence of noncardiogenic pulmonary edema
Unexplained respiratory illness resulting in death, with autopsy showing noncardiogenic pulmonary edema without an identifiable cause
Diagnosis of hantavirus pulmonary syndrome indicated in the health record or hantavirus pulmonary syndrome listed on the death certificate
Laboratory criteria
Detection of one or more of the following:
Hantavirus-specific IgM or increasing hantavirus-specific IgG titer
Hantavirus-specific RNA
Hantavirus antigen on immunohistochemical analysis of lung biopsy or autopsy tissues

* Shown are surveillance criteria from the Centers for Disease Control and Prevention.[22] Surveillance case definitions are not intended for clinical diagnosis.

RIBAVIRIN

Ribavirin is a nucleoside analogue that has been used in the treatment of many viral infections, including Lassa fever, hepatitis C, respiratory syncytial virus infection, and measles, and has shown antiviral activity against ANDV.[32] Ribavirin resulted in dose-dependent inhibition of viral replication in vitro and prevention of hantavirus pulmonary syndrome in animal models when administered early and as postexposure prophylaxis.[30] However, no high-quality clinical trial of ribavirin has shown efficacy in humans with ANDV infection or hantavirus pulmonary syndrome.[33-35] The most methodologically rigorous trial — a placebo-controlled, double-blind trial of intravenous ribavirin in North American hantavirus pulmonary syndrome (caused by Sin Nombre virus) — showed no significant difference in survival or need for ECMO between ribavirin and placebo and was terminated early for futility.[36] Some small case series in Argentina have suggested that early administration of ribavirin during the prodromal phase of ANDV infection may be associated with reduced progression to the cardiopulmonary phase. These preliminary findings underscore the need for controlled clinical trials.[36,37]

OTHER THERAPEUTICS AND INVESTIGATIONAL AGENTS

Other therapies under investigation for treatment of ANDV infection include convalescent plasma, monoclonal antibodies, favipiravir, baloxavir, molnupiravir, RNA interference approaches, and host-directed therapies. Monoclonal antibodies (JL16 and MIB22) that target ANDV glycoproteins showed promise in hamster models, and favipiravir (an RNA polymerase inhibitor) led to a clinically significant reduction in viral load and increased survival in preclinical testing.[8,38-45] One nonrandomized trial suggested that convalescent plasma reduced mortality by close to 50% as compared with comparator cases.[38]

To date, no clinical trials assessing the efficacy or safety of monoclonal antibodies, immune plasma, or small-molecule agents for ANDV infection or hantavirus pulmonary syndrome in humans are underway or have been completed[45]; all advanced therapeutic candidates remain in preclinical or early investigational stages. Additional studies, including randomized, controlled trials of these agents, are urgently needed.

KEY POINTS

ANDES VIRUS

- Andes virus (ANDV) is the sole orthohantavirus with demonstrated human-to-human transmission and has a prolonged incubation period of up to 6 to 8 weeks.
- ANDV causes hantavirus pulmonary syndrome, a disease with a high case fatality rate (25 to 40%) that rapidly progresses from a febrile illness to severe respiratory failure and shock within hours to days.
- Clinicians should maintain a high index of suspicion for ANDV in any patient presenting with fever and severe rapidly progressive respiratory illness in conjunction with a relevant travel or exposure history; early diagnostic clues include marked leukocytosis, thrombocytopenia, and the presence of immunoblasts.
- No licensed targeted antiviral therapy or vaccine currently exists; management is focused on early recognition and aggressive supportive critical care.
- Strict infection prevention and control measures are essential to reduce the risk of transmission and include prompt isolation, use of appropriate personal protective equipment, and urgent public health notification.

VACCINES AND POSTEXPOSURE PROPHYLAXIS

In the first hantavirus pulmonary syndrome vaccine trial involving humans that has shown promise (a phase 1 trial), an intramuscular ANDV DNA vaccine was safe and elicited robust neutralizing antibody responses in healthy volunteers that were sustained for over 330 days.[9] Other vaccine platforms (inactivated virus vaccines and viral vector vaccines) remain in preclinical development. Some experts recommend ribavirin for ANDV postexposure prophylaxis, but this recommendation is not based on solid evidence.[46]

INFECTION PREVENTION AND CONTROL PRACTICES

Primary prevention of ANDV infection involves the elimination of exposure to rodent vectors and their excreta and the use of environmental hygiene measures and PPE to reduce risk.[4] Rodent-infested areas should be cleaned with bleach-based disinfectants by personnel who are using PPE, including N95 (or higher-level) respirators and eye protection, under well-ventilated conditions.[47] Given the risk of human-to-human transmission, infection prevention and control measures are fundamental to the care of patients with ANDV infection.[4] The CDC advises admission of patients to an airborne-infection isolation room and use of a gown, gloves, eye protection, and an N95 or high-level respirator.[47] Although most recognized human-to-human transmission events occur after close or prolonged contact and health care–associated transmission appears to be uncommon, superspreader events and uncertainty about the timing and route of infectiousness support a layered, risk-based approach to infection prevention and control. In Chile, droplet precautions are recommended during routine care of suspected or confirmed cases, with airborne precautions (e.g., use of N95 or level 2 filtering facepiece [FFP2] respirators) reserved for aerosol-generating procedures.[48] In Argentina, current national guidance recommends respiratory isolation, including isolation of patients in a single room, use of N95 or FFP2 respirators while in the room, use of additional PPE to prevent exposure to secretions or fluids, and use of N100 or FFP3 respirators during invasive airway procedures.[49,50] It is important to note that nosocomial transmission of ANDV has been documented but remains uncommon and that most human-to-human transmission events have occurred among close or household contacts.[4,6,15,50,51]

CONCLUSIONS

ANDV is an important emerging pathogen that is capable of causing rapidly progressive disease and high mortality. Clinicians should maintain a high index of suspicion in any patient who presents with fever and a severe, rapidly progressive respiratory illness and who has had exposure to the virus or a history of travel to a region where ANDV is endemic. Rapid onset of leukocytosis, an increased immunoblast percentage, and thrombocytopenia are important early clues. Notification to public health authorities to coordinate testing along with isolation of suspected and confirmed cases is essential. The cornerstone of treatment remains largely supportive and includes prompt, appropriate care with circulatory support in the ICU for hantavirus pulmonary

syndrome; circulatory support may include veno-arterial ECMO. Investigational medical countermeasures are in development and should be administered in research settings only. Infection prevention and control measures are crucial in reducing community and persistent transmission of the disease.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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