IPS Journal of Basic and Clinical Medicine
ISSN: 3115-5731
IPS J Basic & Clin Med, 2(4): 115-121 (2025)
DOI: https://doi.org/10.54117/ijbcm.v2i4.21

Hantaviruses, Transmission Dynamics, Clinical Outcomes, and Preventive Approaches: A Review

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Abstract

Hantaviruses are emerging zoonotic pathogens with a significant global impact, causing severe diseases such as Hemorrhagic Fever with Renal Syndrome (HFRS) and Hantavirus Cardiopulmonary Syndrome (HCPS). These viruses are primarily transmitted to humans through contact with aerosolized excreta from infected rodent reservoirs. The global distribution of Hantaviruses is influenced by environmental factors like climate change, which affect rodent population densities. Pathogenesis is characterized by increased vascular permeability and acute thrombocytopenia, leading to high mortality rates. The virus possesses a tripartite, negative-sense RNA genome, encoding a nucleocapsid protein, glycoproteins, and an RNA-dependent RNA polymerase, which are crucial for replication and host cell entry. Diagnosis of acute infection is primarily achieved through serological tests detecting IgM and IgG antibodies, with molecular methods like RT-PCR providing confirmation by identifying viral RNA. Currently, no specific antiviral treatment exists; management is supportive, focusing on intensive care for respiratory or renal failure. Preventive strategies are paramount and emphasize public education on rodent control, safe cleaning practices in infested areas, and the use of disinfectants. Research into vaccines, including DNA-based platforms, offers promise for future prevention. Given their high fatality rate and potential for person-to-person transmission, Hantaviruses represent a critical and ongoing public health challenge that requires continued surveillance, improved diagnostics, and effective therapeutic interventions.

Keywords: Hantavirus, zoonotic diseases, pathogenesis, prevention

Article History
Received: 10 Sept 2025
Accepted: 26 Sept 2025
Published: 07 Oct 2025

Scan QR Code to view[1]

How to cite this paper: Iheukwumere, I. H., Iheukwumere, C. M., Unaeze, B. C., Ike, V. E., Nnadozie, H. C., & Onyema, S. O. (2023). Hantaviruses, Transmission Dynamics, Clinical Outcomes, and Preventive Approaches: A Review. *IPS Journal of Basic and Clinical Medicine*, *2*(4), 115–121. https://doi.org/10.54117/ijbcm.v2i4.21.

Introduction

According to Calisher et al., in 2007, Hantavirus infection is the most widespread zoonosis that is emerging partially due to global warming, intense rainfall, and increased severity of floods resulting in an annual incidence of about 150000-200000 cases (Malecki et al., 2008). The mode of transmission and circulation of Hantavirus can be influenced by climate change which can impact the population densities of the reservoir host i.e. rodents (Khingstrom et al., 2002).

Rodents' makeup 42% of the total mammalian biodiversity in the world, consisting of 2,277 species that inhabit every continent except Antarctica. They serve as carriers for a diverse range of infectious agents (Malecki et al., 2008). People can get infected with Hantaviruses by breathing in tiny particles of dust that have been contaminated by rodent droppings or urine (Ahlm et al., 2000). The particles lead to various types of organ damage due to a temporary increase in pro-inflammatory cytokines, also known as a "cytokine storm" (Khaib et al., 2002).

Small mammals are the exclusive carriers of bunyaviruses, which are zoonotic agents capable of inducing Hemorrhagic Fever with Renal Syndrome (HFRS), Hantavirus Pulmonary

Syndrome (HPS), or Hantavirus Cardiopulmonary syndrome (HCPS); both of which exhibit case fatality rate up to 50% (Yu et al., 2014). The exact cause of the disease is not well known, although it is believed that both interfere with blood vessels and strong responses from cytotoxic lymphocytes give rise to the development of the symptoms (Lee et al., 2012).

This condition frequently occurs when a person comes into contact with mouse feces or urine within 1 to 3 weeks after the start of symptoms (Escutenaire et al., 2000). Hantavirus infections pose a high mortality rate. It is also worth noting that Hantavirus can be transmitted from person to person, underlining the significance of medical interventions for preventing and treating Hantavirus infections (Lee et al., 2012). Currently, there are more than 28 known Hantaviruses that can cause various diseases in humans globally.

These illnesses can range from renal dysfunction to fluid overload in the lungs and major bleeding conditions (Chen et al., 2011).

Origination of Hantaan Virus

According to Hjelle and Yates, In 1978, the etiologic agent of Korean Hemorrhagic fever was isolated from small infected field rodent (mice) Apodemus agrarius near Hantan river in South Korea. The virus was named as Hantaan virus, after the name of the river Hantan. This initial discovery dates back to scientific approaches that were initiated after the Korean war (1951-1953) (Malecki et al., 2008), during which more than 3000 cases of Korean hemorrhagic fever were reported among UN troops (Leighton et al., 2003).

In 1981, Hantaan virus strain 76-118, isolated from Apodemus agrarius was grown in A549 cell line, and its electron microscopic images revealed that the virus was a new member of the Bunyaviridae family (Armien et al., 2013). It was observed that Hantaviruses unlike other members of this family do not have an arthropod vector, and exclusively establish a persistent infection in the population of their specific rodent hosts. In 1981, a new genus termed as "Hantavirus" was introduced in the Bunyaviridae family (Malecki et al., 2008), which included the viruses that cause hemorrhagic fever with renal syndrome (HFRS). It was initially thought that pathogenic Hantaviruses are restricted to old word (Ahlm et al., 2000).

Until 1993, the only native Hantavirus found in new word was non-pathogenic Prospect Hill virus (PHV) (Chen et al., 2001). This myth ended after the Hantavirus outbreak in the four-corner region of Southwestern United States that caused serious respiratory distress in infected patients and lead to the discovery of a new Hantavirus disease called hantavirus cardiopulmonary syndrome (HPS) (Botten et al., 2000). An examination of frozen stored samples of lung tissue from people who had died of unexplained lung disease in the past revealed that HPS is an old disease with conformed cases dating to at least 1959 (Khaib et al., 2002).

Within a very short period the virus causing HPS was isolated from common dear mouse (Peromyscus maniculatus) and was later named as Sin Nombre virus (SNV) (Yu et al., 2014). Later on, it became clear that other Hantaviruses similar to SNV, such as Andes virus (ANDV), are present through-out the united-states (Calisher et al., 2007). Currently, the Hantavirus genus includes more than twenty-one species and more than 30 genotypes (Botten et al., 2000).

Genome Nature

According to Khingstrom et al., 2002, Electron microscopy reveled that Hantaviruses are spherical or oval particles with a diameter of 80 to 210 nm. They have tripartite negative sense RNA genome. The large (L) segment genomic RNA encodes viral RNA dependent RNA polymerase (RdR), the medium size (M) segment encodes viral glycoprotein precursor (GPC) which is later cleaved into two glycoproteins G1 and G2, and the small (S) segment encodes the viral nucleocapsid protein (N) (Yang et al., 2004). The nucleotide sequence at 5′ and 3′ termini of each genome segment is complementary and undergoes base pairing to form panhandle structures. Inside the virus particle the three genomic RNAs are complexed with N protein and form three individual nucleocapsids, which along with RdRp are packaged within a lipid envelop (Botten et al., 2000). Two glycoproteins G1 and G2 remain embedded within the lipid envelop (Malecki et al., 2008).

According to Calisher et al., in 2007, explaining the activities in Fig 3 frame (a-e) below;

a. Hantaviral genome comprises of three negative sense RNAs, S segment encodes nucleocapsid protein (N), M segment encodes glycoproteins G1 and G2, and L segment encodes viral RdRp (Malecki et al., 2008).

b. b. Panhandle structures of three hantaviral genomic RNAs are formed by the base pairing of complementary bases at 5′ and 3′ terminus of each genome segment.

c. c. Pictorial representation of Hantavirus particle, showing three nucleocapsids enveloped in a lipid bilayer (Yu et al., 2014).

d. d. Thin-section electron micrograph of Sin Nombre virus isolate, a causative agent of Hantavirus pulmonary syndrome (HPS) while e is the pictoral representation of Hantavirus lifecycle (De-arrauyo et al., 2012).

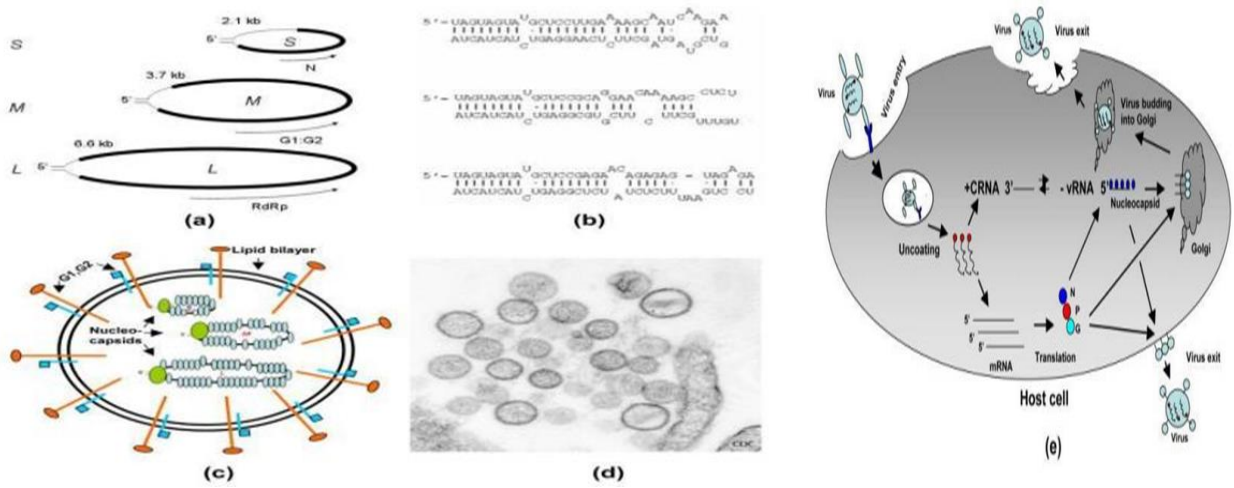

Figure 1: Genome sequence, Hantavirus.
Source: Ahlm et al., 2000.

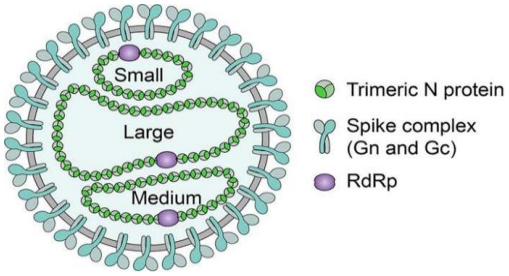

Figure 2: Structure of the Hantavirus
Source: Escutenaire *et al.*, 2000.

Hantaviruses are enveloped with a lipid bilayer containing Glycoprotein spikes assemblies comprised of Gn and Gc Glycoproteins. Contained within the envelope are the equimolar amounts of N Protein packaged S (small), M (medium), and L (large) segments vRNA, which are associated with an RdRp (Botton *et al.*, 2000). SNV structure is generally spherical with a dense envelope (Ahlm *et al.*, 2000). N Protein forms trimers that selectively encapsidates the negative-sense vRNA into RNPs and assists in its panhandle formation for packaging (Hjelle and Yates, 2001).

Table 1: Classification of Hantavirus

Classification	Nomenclature
Kingdom	Virus
Class	Elliviricetes
Family	Bunyaviridae
Sub-Family	Hantaviridae
Genus	Orthohantavirus
Species	Hantavirus
Vernacular name	Old World virus (Hokkaido virus Hokv, Muju Virus Mju); New World virus (Sin Nombre virus SNV, Andes virus ANDV)

Source: Leighton *et al.*, 2003.

Structural Proteins and their Functions

Nucleocapsid protein (N)

According to Armien *et al.*, 2003, N protein is the most abundant Hantavirus protein found in the cytoplasm of infected cells. Its transcript is detected in infected cells six hours post infection. N protein is responsible for encapsidation and packaging of the viral genome. However, recent studies have shown that N is a multifunctional protein involved in diverse viral functions, including its role in the transcription and translation initiation of viral mRNA (Yang *et al.*, 2004). Trimeric N also recognizes vRNA panhandle with specificity and likely facilitates the selective incorporation of viral genomic RNA into virions. N has also been found to interact with multiple host cell proteins and the nature of such interaction remains unclear (Clement *et al.*, 2014).

Glycoprotein

Glycoprotein precursor (GPC) is synthesized on ribosomes associated with endoplasmic reticulum (ER) and is translocated to ER lumen by an endogenous signal peptide (Yang *et al.*, 2004). In ER, GPC is post translationally cleaved at a conserved WAASA site, and two glycoproteins G1 and G2 are generated which are later glycosylated and translocated to the Golgi. Glycoproteins facilitate the attachment of virions with the integrin receptors located on the host cell surface (Armien *et al.*, 2013).

RdRp

Hantavirus RdRp is a huge protein with a molecular weight of 250-280 KD (Yu *et al.*, 2014). Because of its large molecular weight RdRp is difficult to express in bacteria, and thus remains the most uncharacterized protein in Hantaviruses. RdRp mediates both transcription and replication of viral genome. During transcription RdRp synthesizes viral mRNA from negative sense vRNA template (Hjelle and Yates, 2001). During replication RdRp replicates vRNA genome via a cRNA intermediate. Thus it is likely that Hantavirus RdRp has multiple activities, including endonuclease, replicase, transcriptase and RNA helix unwinding activities (Yang *et al.*, 2004). However, recent studies have shown that viral RdRp requires N protein for function (Calisher *et al.*, 2007). For example, short capped primers generated from host cell mRNAs by the process of cap snatching are used by RdRp to initiate the transcription. Hantavirus N protein has been

found to be involved in the generation of such capped primers (Lee *et al.*, 2012).

Distribution of the Disease

Currently it is estimated that 150,000 to 200,000 cases of Hantavirus disease occur each year around the world (Yu *et al.*, 2014), with the majority being reported in Asia such as China, Korea, Eastern part of Russia and some countries within Europe like Russia, Finland and Sweden during the winter season (very cold climate) (Lee *et al.*, 2012). Majority of Hantavirus patients are males with age of 20 to 50 years who either have Mice as pets, farmers who train mice or cleaners who gets the virus while cleaning mice faeces. Mortality rate depends upon the type of disease with their respective Hemispheres (Chen *et al.*, 2011).

Properties of the Virus

Physical properties

i. Hantaviruses are enveloped RNA viruses (Khingstrom *et al.*, 2002)
ii. Spherical in shape with a diameter of 80 to 120 nm.
iii. They form a separate genus within the Bunyaviridae family (Lee *et al.*, 2012)
iv. iv. There's the presence of a Lipid protein like all other enveloped virus.
v. There's the presence of an extra sheet called capsid in their physical make-up which retracts outward penetration (Ahlm *et al.*, 2000).

Biological properties

i. The genome comprises three negative sense, single-stranded RNAs that share a 30 terminal sequence of the genome segments (Lee *et al.*, 2012).
ii. ii. The three segments, S (small), M (medium) and L (large), encode the nucleoprotein (N), envelope glycoproteins (Gn and Gc), and the L protein or viral RNA-depended RNA polymerase, respectively.
iii. iii. Hantaviruses replicate in the cytoplasm and the glycoproteins are targeted to the Golgi complex, where most Hantaviruses bud (Botton *et al.*, 2000).
iv. iv. Until now, it is considered that b1-integrin interacts with viral Gn ofapathogenic Hantaviruses, while b3-integrin interacts with the glycoprotein of pathogenic Hantaviruses (Khignstrom *et al.*, 2002).

Chemical properties

i. Like other enveloped viruses, Hantaviruses are readily inactivated by heat (30 min at 60C), detergents, UV irradiation, organic solvents and hypochlorite solutions (Ahlm *et al.*, 2000).
ii. Bleach can serve as a useful disinfectant on all areas with the rodent's faece droppings (Leighton *et al.*, 2003).

Pathogenesis of Hantavirus

The pathogenesis of these diseases involves complex interactions between the virus and the host, particularly the vascular endothelium, immune cells, and various molecular mediators, leading to vascular leakage, thrombocytopenia, and organ dysfunction.

Viral Entry and Endothelial Infection

Following inhalation of aerosolized excreta from infected rodents, hantaviruses infect cells of the respiratory tract, then spread to infect vascular endothelial cells, macrophages, and dendritic cells. A critical step in their pathogenicity is the interaction with **β3 integrins**, which mediate attachment and entry into endothelial cells (Gavrilovskaya et al., 1999; Kanerva et al., 1999). Pathogenic hantaviruses such as Hantaan virus (HTNV) use β3 integrin receptors, whereas nonpathogenic strains may rely on β1 integrins (Gavrilovskaya et al., 1999; Sironen *et al.*, 2015; Iheukwumere *et al.*, 2024a).

Endothelial Dysfunction and Vascular Leakage

Although hantaviruses infect endothelial cells, there is minimal cytopathic effect. Instead, the dysfunction arises from altered endothelial cell behavior. Pathogenic hantaviruses have been shown to inhibit β3 integrin–directed endothelial cell migration, which impairs vascular repair and contributes to capillary leakage (Gavrilovskaya et al., 2002; Iheukwumere *et al.*, 2024b). Infected endothelium displays increased permeability in response to permeability-enhancing factors, such as vascular endothelial growth factor (VEGF), which exacerbate fluid leakage into tissues, causing pulmonary edema in HPS or renal interstitial edema in HFRS (Koster & Mackow, 2012; research progress review, 2022).

Immune Response and Cytokine Storm

The immune response plays a dual role. On one hand, it helps control viral replication; on the other, its dysregulation contributes to disease. Infection induces production of type I and III interferons, pro-inflammatory cytokines (e.g., IL-1β, IL-6, TNF-α), and chemokines such as RANTES and IP-10 from infected endothelial cells and immune cells (Saavedra et al., 2021; research progress review, 2022). T cells, especially CD8+ cytotoxic T lymphocytes, and NK cells infiltrate infected tissues and may contribute to immunopathology (Terajima & Ennis, 2011; Iheukwumere *et al.*, 2024c).

Platelets, Coagulation, and Hemorrhagic Manifestations

Another component of hantavirus pathogenesis involves platelet dysfunction and coagulation abnormalities. Platelet binding to infected endothelial cells mediated by β3 integrins has been observed, which may contribute to thrombocytopenia and impaired hemostasis (research progress review, 2022; Gavrilovskaya et al., 1999). Moreover, the suppression of certain endothelial proteins involved in maintaining vascular integrity, such as thrombospondin-1, has been demonstrated in infections with pathogenic hantaviruses (Frontiers study, 2016; Iheukwumere *et al.*, 2024d).

Organ Damage and Clinical Outcomes

In HFRS, renal injury (including tubular damage, proteinuria, hematuria, and acute kidney injury) predominates. In contrast, in HPS, acute pulmonary edema leads to respiratory distress and shock. The severity is often linked more to the host response than direct viral cytopathy, where excessive immune activation, vascular leak, and multiorgan involvement drive

morbidity and mortality (Koster & Mackow, 2012; research progress review, 2022; Iheukwumere *et al*., 2024e).

Mode of Transmission

Hantavirus is transmitted via contact with rodents like rats and mice, especially when exposed to their urine, saliva and inhaling their faces or mouse droppings (Botton *et al.*, 2000).

Replication of Hantavirus

Viral Replication of Hantaviruses

Viral ribonucleoproteins (vRNPs), composed of genomic RNA encapsidated by the nucleocapsid (N) protein and associated with the L protein, serve as the functional units of genome replication and transcription (Calisher et al., 2007). The L protein harbors the RNA-dependent RNA polymerase (RdRp) that catalyzes both replication and transcription (Lee et al., 2012; Iheukwumere *et al*., 2025a).

Once vRNPs are released into the cytoplasm (Chen et al., 2011), replication begins de novo via synthesis of a positive-sense complementary RNA (cRNA), which serves as a template for progeny negative-sense genomic RNA. Like the vRNA, the cRNA is encapsidated by N proteins. Interestingly, although self-initiating polymerases usually use purines as initiating nucleotides, hantavirus replication begins with a uridine monophosphate, suggesting a priming–realignment mechanism with cleavage of the overhang (Malecki et al., 2008; De-Araujo et al., 2012; Iheukwumere *et al*., 2025b).

While vRNA and cRNA are consistently encapsidated by N proteins, viral mRNA typically is not (Yu et al., 2014). However, encapsidation of La Crosse virus (Peribunyaviridae) S segment mRNA with the N protein has been reported, albeit with much lower affinity than vRNA and cRNA (Leighton et al., 2003). Encapsidation of mRNA by N may inhibit its translation, acting as a negative feedback mechanism when intracellular N levels are high (Khaib et al., 2002; Iheukwumere *et al.*, 2025c).

The hantavirus genome contains conserved non-coding untranslated regions (UTRs) at both termini, which are hypothesized to facilitate circularization into a panhandle conformation (Chen et al., 2011; Iheukwumere *et al*., 2025d). More recent structural evidence suggests that both ends of the RNA can instead be bound by the L protein at distinct binding sites, implying that genome circularization seen in electron microscopy may be due to L–RNA interactions, or a combination of base-pairing and protein–RNA contacts (Klingström et al., 2002; Yu et al., 2014).

Following transcription by a cap-snatching mechanism, viral mRNAs are translated (Ahlm et al., 2000). For Hantaan virus (HTNV), it has been proposed that the N protein substitutes for the eukaryotic initiation factor 4F (eIF4F), promoting preferential translation of viral mRNAs (Calisher et al., 2007). Structural studies suggest that HTNV N resembles Programmed Cell Death 4 (PDCD4), a tumor suppressor that interferes with eIF4F assembly, and may thus simultaneously inhibit host protein synthesis (Armien et al., 2013; Iheukwumere *et al*., 2025e).

The precise intracellular site of hantavirus replication and transcription remains under investigation. Evidence shows that HTNV N protein induces the formation of perinuclear structures that colocalize with markers of the endoplasmic reticulum–Golgi intermediate compartment (ERGIC), but only minimally with ER or Golgi markers (Lee et al., 2012). These structures, resembling viral factories, stained positive for vRNA and cRNA and appeared tubular in nature (De-Araujo et al., 2012). Moreover, the recruitment of stress granules to these sites suggests that hantaviruses establish replication factories within remodeled Golgi compartments associated with stress granules (Khaib et al., 2002; Iheukwumere *et al*., 2025f).

Disease of Hantavirus

There is no known disease just the signs and symptoms it elicits in an infected patient.

Clinical Manifestation of the Virus

Signs and symptoms

As stated by Escutenaire *et al.*, in 2000, In most recorded cases, symptoms develop 1 to 8 weeks after exposure. Early symptoms, such as body aches, headaches, diarrhea and abdominal pain while infected patients can also show signs such as dry cough and fever (Yu *et al.*, 2014).

Syndromes

As stated by Armien *et al.*, in 2013, Overall, there are three syndromes of Hantavirus which include the following;

i. Haemorrhagic fever with renal syndrome (HFRS), mainly in Europe and Asia.
ii. Nephropathia epidemica (NE), a mild form of HFRS, caused by Puumala Hantavirus, and occurring in Europe (De-arauyo *et al.*, 2012).
iii. Hantavirus cardiopulmonary syndrome (HCPS), common in the Americas (Botton *et al.*, 2000).

Diagnosis

Physical Examination

With the patient showing signs and symptoms of being infected with the virus, the Blood of the patient is collected and diagnosis done on the Blood sample (Armien *et al.*, 2013).

Sample Collection

The Blood of the suspected patient is collected using a sterile syringe intravenously (Lee *et al.*, 2012).

Serological Test

Laboratory diagnosis of acute Hantavirus infections is based on serology as virtually all patients have IgM and usually also IgG antibodies present in serum at the onset of symptoms (Yang *et al.*, 2004; Iheukwumere *et al*., 2025g). The most commonly used serological tests are indirect IgM and IgG ELISA as well as IgM capture ELISAs, which have higher specificity than indirect ELISAs. Indirect immunofluorescence assays are also regularly used for diagnostics but have lower specificity (Khaib *et al.*, 2002;

Iheukwumere *et al*., 2025h). In addition, rapid 5-minute user-friendly.immune-chromatographic IgM-antibody tests have been developed and are available commercially (Escutenaire *et al.*, 2000; Iheukwumere *et al*., 2025i).

The Hantavirus infection can also be confirmed by detection of Hantavirus genome in blood or serum samples by RT-PCR. Both traditional and quantitative RT-PCR are used to detect viraemia (Botton *et al.*, 2000). Although the presence of viraemia varies, viral RNA can usually be detected if an acute sample is available. It has also been suggested that higher viraemia is found in more severe Hantavirus infections (DOBV, SNV, ANDV), compared with milder infections, caused by PUUV (Leighton *et al.*, 2003; Iheukwumere *et al.*, 2025j). In addition, with detection of viral RNA, Hantavirus infection has been confirmed even before the presence of specific antibodies (Yang *et al.*, 2004).

Treatments

Treatment in the ICU is mostly supportive and may include intubation and oxygen therapy (Escutenaire *et al.*, 2000), fluid replacement and use of medications to support blood pressure. Sometimes antiviral drugs, such as ribavirin, are used to treat other strains of Hantavirus and associated infections (Leighton *et al.*, 2003).

Preventive Measures

i. Put on rubber or plastic gloves (Chen *et al.*, 2011).
ii. Clean up all rodent urine, droppings, nests, or dead mice or rats by using a disinfectant as described earlier.
iii. Mop floors and/or spray dirt floors with disinfectant (Malecki *et al.*, 2008).
iv. Clean countertops, cabinets, and drawers with a disinfectant.
v. Apart from that, only two molecular vaccines against HFRS have been tested in humans, the first was recombinant vaccinia-vectored vaccine expressing the M segment of HTNV and the second plasmid DNA. The advantage of DNA vaccines is that they offer an easy way to construct multivalent vaccines and they are able to induce long-lasting humoral and cellular immunity in Human.
vi. Seek medical care once signs, symptoms are being felt.

Conclusion

Hantaviruses remain a major global zoonotic threat with significant morbidity and mortality, particularly in regions where human–rodent contact is common. The viruses are transmitted primarily through aerosolized excreta from infected rodents and cause severe diseases such as Hemorrhagic Fever with Renal Syndrome (HFRS) and Hantavirus Cardiopulmonary Syndrome (HCPS). Their tripartite, negative-sense RNA genome encodes structural and nonstructural proteins essential for replication, host interaction, and immune evasion. Pathogenesis is largely driven by immune-mediated endothelial dysfunction, leading to increased vascular permeability, thrombocytopenia, and multi-organ damage.

Despite advances in molecular diagnostics, including RT-PCR and serological assays, effective antiviral therapies remain limited, with management relying mainly on supportive care. Preventive strategies—such as rodent control, environmental hygiene, and community education—remain the most effective means of reducing transmission. Promising developments in DNA-based vaccine research offer hope for long-term control and prevention.

Continued surveillance, public health awareness, and research into the molecular mechanisms of Hantavirus infection are crucial for developing targeted therapeutic and preventive interventions. Strengthening diagnostic capacity and investing in vaccine development will be essential to mitigate future outbreaks and reduce the global burden of Hantavirus-related diseases.

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