Hunting Hantavirus: A Quick Guide for Healthcare Professionals before It Invades the Globe unlike COVID-19

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ABSTRACT

A genus of the *Bunyaviridae* family that causes Hantavirus Infections first described during the Korean war. Infection mainly occurs in rodents and humans. It is widely circulated in insectivores and bats. Some of these viruses will quickly infect humans through aerosols or dust produced from infected animal waste products. For humans, clinical cases differ for severity: Some hantaviruses tend to cause mild illness, usually with complete recovery; in the event, some may cause serious illness with fatality. Hantaviruses induce hemorrhagic fever in humans either renal syndrome (HFRS) or cardiopulmonary hantavirus syndrome (HCPS). These illnesses typically develop from 1 or 2 weeks following exposure and are characterized by breathing difficulty, headache, Pneumonia, Abdominal pain, cough, and respiratory failure. This article helps in making mankind alert and proactive.

Key Words: Hantavirus, illness, symptoms, alert.

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INTRODUCTION

Viral infections are considered as one of the principal threats to human life and health worldwide [1, 2]. An ortho hantavirus (hantavirus) in the *Bunyavirales* family is a single-stranded, enveloped, n-sense RNA virus [3]. Humans become contaminated with hantaviruses through interaction with semen, saliva, or feces of rodents [4]. Such strains cause possibly lethal diseases in humans, such as hantavirus hemorrhagic fever with renal syndrome (HFRS) or hantavirus pulmonary syndrome (HPS), also known as hantavirus cardiopulmonary syndrome (HCPS). HPS is an occasional breathing disorder connected with a breath of aerosolized rodent defecations (urine/feces) infected with hantavirus particles [5]. Hantaviruses include Hantavirus species within the Bunyaviridae family. Based on the form of detection the virion shows circular or pleomorphic morphology and has a size of 120–160nm [6]. The virus triggers renal syndrome (HFRS) hemorrhagic fever and even pulmonary hantavirus (HPS) syndrome. HPS diseases have a fast onset of muscle pain and fever which leads to acute breathing suffering [7].

Species Affected

Mice, insectivores, and bats Recognized hantavirus reservoir hosts include mice, insectivores (e.g., shrews, moles), and bats. The growing virus is supposed to be unique to one or more species, although it cannot be uncommon to spill into mouse, insectivore, and bat hosts. Animal members of the Apodemus family bear Hantan, *Amur souchong*, and *Dobrava Belgrade* viruses [8]. Norway rats (*Rattusnorvegicus*) are important reservoir hosts for the Seoul virus but this virus was also found in other rat organisms, including *R. ratus, R. losea, and R. nitidus*. Deer mice (*Peromyscus maniculatus*) bear Sin

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Nombre virus, while in cotton rats (*Sigmodon hispidus*) [9]. Black Creek Canal and Muleshoe viruses were identified. Andes virus and its variations exist in rodents fitting to Akodon and Necromysof South American mouse and the genus Oligoryzomys of rice rat. The Laguna Negra virus was found in Calomyslaucha and Calomyscallidus, while Oligoryzomys was transmitted by Rio Mamore. Some hantaviruses were also identified in laboratory rats and rodents, and pet rats have been diagnosed with the Seoul virus [10]. In numerous laboratory animals, including rats, mice, and hamsters, experimental infections were found. Shrews and moles are bearing quite a few hantaviruses. The presently identified or suspected disease-causing viruses include the *Thottapalayam virus* that infects an Asian musk shrew, Suncusmurinus; Bowe virus found in an African musk shrew, Crociduradouceti; and Uluguru virus reported in the Geat mouse shrew (*Myosorex geata*). Bats bear their hantaviruses but in these species [11], Hantavirus and *Andes virus* (variant *Araraquara*) have also been identified. As of 2018, no bat-associated hantaviruses were detected in animal or human clinical events.

Genome

Like other *Bunyavirales* members, ortho-hantaviruses are enclosed viruses with a genome consisting of 3 single-stranded, negative-sense sections of RNA called S (small), M (medium) and L (large). The S RNA encrypts the enzyme nucleocapsid (N). The M RNA codes a polyprotein that is cleaved cotranslationally to produce the membrane of glycoproteins Gn (formerly G1) and Gc (formerly G2). The L RNA codes the L protein, which acts as the viral transcriptase/replicate [12]. Within virions, hantavirus genomic RNAs are assumed to be complex to form helical nucleocapsids with the N protein, the RNA part of which circulates due to arrangement between the 5 'and 3' terminal sequences of genomic segments with other Bunyavirales, each of the 3 sections has a 3'-terminal nucleotide sequence consensus (AUCAUCAUC), which is harmonizing to the 5'-terminal arrangement and is separate from that of the other 4 genera in the genus [13]. Such sequences seem to create a panhandle arrangement that is likely to play a role in replication and encapsulation through connecting with the viral nucleocapsid (N) protein. The wide section is 6530–6550 nucleotides) (in length, the medium is 3613–3707nt in length and the tiny one is 1696–2083nt in length. Like other genera in this genus, no non-structural proteins are identified. There are brief noncoding intervals at the 5 'and 3' of each line: the noncoding line at the 5 'end of both intervals is 37–51nt. Non-coding regions of 3 'differ: segment L 38–43nt; segment M 168–229nt; segment S 370–730nt. The 3 'end of the S segment is held between the genera which indicates a functional position [14].

Hantavirus N protein

The protein Hantavirus N is of about 433 amino acid residues (about 50 kDa in size). Between different hantaviruses, the N protein seems to be highly conserved. High quantities of N protein are released early after infection. Initial immune retort in Hantavirus patients has also been found to be mainly guided toward N Protein. Hence, several virus diagnostics produced are focused on the identification of Hantavirus N protein or anti-N antibody-protein [15]. The N protein is found solely inside the contaminated cell's cytoplasm. Hantavirus N protein plays a crucial role in the life cycle of the virus as it is necessary to capture viral RNA and to control the replication of viruses and assemble N Protein. The S-segment-encoded N protein is a non-glycosylated protein given a molecular weight of 50 kDa. Both the N protein and RdRp are required for replications, to have an adequate pool of the N protein, mRNA transcription and translation are supposed to precede replication initiation and the concentration of free N protein is supposed to mediate the change from mRNA amalgamation to replication [16]. In comparison, N protein trimers bind to the preserved panhandle structures that are found only in complementary RNA (cRNA) and vRNA, rendering the mRNA naked. Recent reports of the N protein being responsible for sequestering the primers carrying a 59-cap, a crucial prerequisite for efficient translation in eukaryotic cells, from cellular processing (P) bodies suggest even more direct involvement of N protein in virus replication. Immunofluorescence staining of hantavirus-infected cells by N protein antibodies or acute phase HFRS-patient sera is characterized by a granular staining pattern [17]. This staining is suggested to be either due to aggregation of the N protein to inclusion bodies or due to the accumulation of P bodies. The N protein also localizes to the perinuclear region and is membrane-associated, albeit devoid of transmembrane (TM) helices.

Hemorrhagic Fever with Renal Syndrome

HFRS typically shows moderate to serious kidney-related symptoms. The phase of the illness was classically categorized into febrile, hypotensive/proteinuric, oliguric, diuretic, and convalescent periods. For severe illness, these signs are typically more apparent and observed in minor cases. HFRS normally begins off suddenly. Fever, chills, prostration, vomiting, and backache may be the main health symptoms. Gastrointestinal symptoms can also be identified like diarrhea, vomiting and stomach discomfort; in certain instances, the discomfort can be extreme enough to cause appendicitis [18, 19]. Many non-specific health symptoms can also be visible, such as inserted mucous membranes, photophobia, a swollen face, and conjunctivae, or a petechial rash that normally appears on

the palate or neck. In certain situations, even transient vision disability (e.g., decreased visual acuity) exists. Usually, the prodromal stage lasts for a few days to a week, accompanied by the emergence of renal signs. The first step is the step of the proteinuric activity. In this time, hypotension can develop and may last for hours to days. Nausea and vomiting are normal, and acute shock may trigger death. In extreme HFRS situations, a usually oliguric period follows the proteinuric level, then a diuretic/polyuric process as kidney function improves. Death may occur at any phase, but the hypotensive or oliguric stages are especially frequent. In serious cases, kidney failure may occur. Many HFRS may have contact with the lungs, usually to a smaller degree than in HPS. In other instances, it is restricted to mild pulmonary symptoms or X-ray anomalies (particularly pleural effusion); Significant symptoms, including pulmonary edema and reduced pulmonary activity, are also likely. Neurological symptoms, like meningoencephalitis, or clinical indications linked to different other organs (e.g., proof of liver involvement) can occur periodically. Thrombocytopenia is normal and, particularly in more extreme situations, hemorrhagic symptoms like petechiae, hematuria, or melena can be shown there is scope for disseminated intravascular coagulation. Total recovery can take weeks or months but regular kidney function is typically restored by patients. Some training has designated that certain individuals may suffer sequelae from chronic renal dysfunction and hypertension [20]. In a few instances, the irreversible neurological injury was identified.

Hantavirus Pulmonary Syndrome (HPS)

HPS is present in North, Central and South America. HAS is a pulmonary disorder and is sometimes lethal. The causative factor in the United States is the Sin Nombre virus which is borne by deer mice. Symptoms of the illness comprise flu-like indications such as fatigue, cough, abdominal discomfort, headache, and lethargy. It is distinguished by a sudden occurrence of the smallness of sniff with increasingly developing pulmonary edema and is frequently lethal following mechanical ventilation and active diuretics intervention [21]. The mortality rate is 36%. HPS was first identified in the Four Corners area of the southwestern United States after the epidemic of 1993. Dr. Bruce Tempest named it. It was initially named "Four Corners fever," but after protests from Native Americans that the word "Four Corners" stigmatized the area, the term was modified into "Sin Nombre virus", and spread across the U.S. The primary protection remains pest management inside and outside the house. Diagnosis of HPS in a person that has been sick for just a few days is complicated since early signs such as cough, body aches, and tiredness are often confused for influenza. Nevertheless, if the individual experiences fever and fatigue, and has a history of potential exposure to rural rodents along with shortness of breath, it will be strongly predictive of HPS. If the individual has experience. There is no clear hantavirus diagnosis, cure or vaccine. We do note, however, that if infectious people are detected early and seek medical attention in an intensive care unit, they will do better. Patients are incubated in intensive care and Oxygen therapy provided to support them through extreme respiratory distress. The patient goes in intensive c earlier. Pulmonary signs occur in HPS. This condition is often initially characterized by a non-specific illness, typically lasting 3 to 5 days, comparable to the HFRS prodromal level respiratory discomfort and hypotension typically manifest suddenly, accompanied by cough and tachypnea, pulmonary edema, and hypoxia. There might also be heart irregularities, such as bradycardia, ventricular tachycardia or fibrillation. Patients may deteriorate rapidly after the start of the cardiopulmonary phase; some will need mechanical ventilation within 24 hours. Thrombocytopenia is widespread and can begin as early as the stage of prodromal growth. Hemorrhagic symptoms tend to be uncommon in North American patients with HPS but are recorded more often in South America. This is likely to have kidney injury but it appears to be moderate. The Andes, Bayou, and Black Creek viruses tend to be more general. Neurological symptoms have seldom been recorded. While healing is swift and patients typically regain complete lung capacity, it may take weeks to months for convalescence.

SIGN AND SYMPTOMS

The "incubation period" is not recognized favorably, owing to the low number of HPS events. Based on minimal knowledge, however, it appears that symptoms can grow within 1- 8 weeks of exposure to fresh feces, droppings, or saliva of contaminated rodents [22].

Early Symptoms

Early signs include weakness, nausea, and muscle aches, especially in the broad groups of muscles, thighs, knees, back, and occasionally shoulders. These are common signs. Headaches, dizziness, chills and stomach issues can also arise, such as nausea, vomiting, diarrhea, and abdominal pain. Around half of all patients with HPS feel those symptoms.

Late Symptoms

Late HPS signs occur 4-10 days after the initial period of illness. Those involve nausea and smallness of sniff and the sensation in the lungs.

CONTROL AND PREVENTION

The fast recognition of virus-specific 'danger signals' and the activation of both innate and adaptive immunity are needed for successful host defense against viral pathogens [23, 24]. The most successful way to monitor hantaviral diseases is by rising human access to and excrement from contaminated rodents. To study the occurrence of hantavirus in rodent populations may give some warning of an expected increase in the numbers of human cases. Centers for Disease Control and Prevention (CDC) guidelines advocate for rodent inspection of dwellings, reduction of rodent cover around buildings, minimization of rodent food, trapping in and around buildings and correct removal of dead rodents. Removal of rodents from a non-rodent-proofed ranch building did not reduce rodent infestation, while the application of simple rodent-proofing measures to dwellings. Similarly, workplaces and conditions in agriculture, forestry, and military activities should be modified when possible to reduce human-rodent exposure. In China, a robust prevention policy against hantavirus infection (HVI) has been introduced in the most resistant regions, including environmental awareness and promotion, pest management, monitoring, and a vaccine. Eradicating the lake hosts of hantaviruses is neither reasonable nor attractive as of the spacious circulation of sigmodontine rodents in North America and their meaning in the job of organic ecosystems. The paramount now untaken slant for disease influence and prevention is stake fall through environmental modification and cleanliness practices that deter rodents from colonizing the home town and go to a work environment, as acceptably as a nontoxic attack of rodent leftover and nesting materials. prohibited experiments carry out demonstrated that plain and cheap methods are useful in preventing rodents from inward bound rural dwellings. Such guidelines emphasize avoidance of HPS in the Americas along with sigmodontine rodents. While the possibility of contracting hantavirus disease from native violin rodents in North America or through the introduction of murine rodents in the Americas is small, the human pathogenicity location of each hantavirus settled upon by these rodent classes has not been established [25]. The precautions described in this bang are broadly applicable to the complete groups of rats and mice.

EPIDEMIOLOGY

Infections with Hantavirus are related to local, job-related or recreational practices, typically in rural settings, which bring people into interaction with diseased rodents. Established human hantavirus infections mainly occur in adults. HPS cases occur during the year in the United States but most are recorded in spring and summer. HVI (a consequence of HPS or HFRS) was epidemiologically related to the following situations [26]:

- The rising amount of host mice residing in human dwellings
- Inhabiting or scrubbing formerly bare cabins or other persistent rodent-infested dwellings
- Sheds and other outhouses were cleaned
- Troubling defecations or rodent dens in the house or workplace
- treating mice without gloves
- Holding wild rodents in captivity as livestock or test subjects
- Handling tools or machines in storage
- disrupting excreta in rodent-infested areas during walks or campsites
- sleeping on the ground
- Tilling by hand or planting

RISK FACTORS

Hantavirus pulmonary syndrome is nearly all regular in pastoral areas of the western United States through the bound and summer months. Other hantaviruses arise in Asia, everywhere they produce kidney disorders to a certain extent than lung problems. The coincidental of mounting hantavirus pulmonary syndrome is larger for fill with who work, live or amuse yourself in chairs everyplace rodents live. Factors and actions that step up the threat include [27]:

- Opening and cleaning slow unused buildings or sheds
- Housecleaning, acutely in attics or other low-traffic areas
- Having a household or workspace diseased with rodents
- Having a task that involves exposure to rodents, such as construction, function creation, and mosquito control
- Camping, mountaineering or hunting

CONCLUSION

Hantavirus causes severe respiratory disturbances followed by hypertension and sudden death. So, cleanliness and hygienic conditions to be maintained while handling experimental animals. And utmost care to be taken while disposing of the waste of the animals. The health care professional should know about this before it invades the globe, unlike COVID-19.

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Conflicts of Interests

Authors do not have any conflicts of interest with the publication of the manuscript.

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