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HANTAVIRUS - "A RODENT BORN KILLER DISEASE" - A REVIEW

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ABSTRACT

Hanta viruses are emerging rodent borne viruses that cause hemorrhagic fever in human. The symptoms are enhanced vascular permeability, thrombocytopenia and plasma leakage. One of the target organs of hanta virus induces hemorrhagic fever with renal syndrome and an infection often results in acute renal failure. The diagnosis rests on serologic evidence. Maintenance of BP and treatment of renal or respiratory insufficiency is indicated. The lack of vaccines and a specific antiviral therapy renders the prevention of infection as the only way to decrease the emergence of Hantaviruses cases. This paper reveals about the hantavirus infection and its effects on kidney in brief.

KEY WORDS

Hanta virus, Thrombocytopenia, Hemorrhage, Vaccines and Antiviral therapy.

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INTRODUCTION

The old world hantaviruses, members of family bunyaviridae, cause hemorrhagic fever with renal syndrome (HFRS), transmission to humans occurs via inhalation of aerosols contaminated with the excreta of infected rodents. The viral antigen is detectable in dendritic cells, macrophages, lymphocytes, and most importantly, micro vascular endothelial cells[1]. However, the site and detailed mechanism of entry of HFRS-causing hantaviruses in polarized epithelial cells have not yet been defined. In contrast to other genera of the family Bunyaviridae, these viruses infect humans when hantavirus - contaminated excretions from

persistently but asymptomatically infected rodents are inhaled [2, 3]. Hantaviruses are enveloped viruses with a single-stranded negative-sense RNA consisting of three segments. The small (S) segment encodes the nucleocapsed (N) protein. The medium (M) segment encodes the two envelope proteins (Gn and Gc), and the large (L) segment encodes the viral RNA polymerase (so), within the genus Hantavirus, two groups have been identified: New World and Old World hantaviruses. They not only differ in their geographical distribution but also vary regarding the pathology of human infection. New World hanta viruses are the causative agent of hemorrhagic fever with renal syndrome (HFRS). Renal manifestation includes acute tubulo interstitial nephritis and interstitial hemorrhage, leading to acute renal failure. Severe forms of HFRS may even result in chronic renal failure. Despite differences concerning the main target organ, a common characteristic of both HPS and HFRS infection is capillary leakage to infection of vascular endothelial cells[4].

Defintion

Hantavirus is a virus that is transmitted by rodents and causes infection in humans.

Types

It causes tow distinct human diseases; hemorrhagic fever with renal syndrome (HFRS) and human pulmonary syndrome (HPS). The causative agent, Hantaan virus (HTNV) was identified in 1978, until now 21 different hantavirus species have been, and more than 30 genotypes are characterized. The genus hantavirus is roughly composed of two main groups; old world and new world[5].

Hantaviruses in humans is called by pathogenic old world hantaviruses that include Amur virus, Seoul virus, HTNV, as Dobrava virus. (DOBV), Tula virus (TULV), and puumala virus (PUUV) [6].

Morphology

Hantaviruses comprise one of five genera of the virus family Bunyaviridae[7]. They replicate in the cytoplasm of host cells and are composed of a spherical lipid envelope; four viral proteins; and three single-stranded, negative-sensed RNA segments designated S (small), M (medium), and L (large) that are coding for the nucleocapsi protein (NP), the surface envelope glycoprotein's G1, and G2, and the RNA polymerase, respectively[8].

Epidemiology

More than 3000 United Nations and US soldiers experienced an acute febrile illness with acute renal failure and shock and a mortality rate of 7% close to a small river called Hantaan. The first pathogenic New World hantavirus (Sin Nombre virus) was discovered in the early 1990s in the Four Corners region of the United State[9]. From this time on, numerous additional pathogenic New World hantaviruses were identified and characterized (Table No.1). New World hantaviruses are the causative agent of approximately 300 cases of HPS each year in North and South America, with lethality rates up to 50%[9].

Pathogenesis

Hantavirus replication takes place in macrophages and vascular endothelial cells, especially in the lung and the kidney, the entry into host cells occurs by attachment to a integrin on the cellular surface and subsequent endocytosis[10].

The viral RNA-dependent RNA polymerase transcriptions of viral genes and replication of the ciral genome segment. The viral NP and RNA polymerase mRNA are translated at free tibosomes, whereas the glycoprotein mRNA is translated into the endoplasmic reticulum G1 and G2 glycoprotein's are transported to the Golgi complex for final glycosylations. Large intracellular inclusion bodies, probably composed of NP, are form the cytoplasm[11]. It is assumed that hantavirions are formed at the membranes of the Golgi complex, followed by budding into the Golgi cisternae, migration in secretary vesicles to the plasma membrane, and release by exocytosis[12].

Causes

The main natural reservoir of Hantaviruses is murid rodents. Studies have reported Hantavirus infections to be present in animal species other than rodents, cattle, moose, cat, and dog[13].

Symptoms

Symptoms of HFRS usually develop within 1 to 2 weeks after exposure to infectious material, but in rare cases, they may take up to 8 weeks to develop.

Initial symptoms begin suddenly and include intense headaches, back and abdominal pain, fever, chills, nausea, and blurred vision. Individuals may have flushing of the face, inflammation or redness of the eyes, or a rash. Later symptoms can include low blood pressure, acute shock. Vascular leakage, and acute kidney failure, which can cause severe fluid overload[14].

Diagnosis

Hantavirus diagnosis should be performed in a patient with fever, lumbago, renal failure, and recent outdoor activities. In the early course of the disease, thrombocytopenia is detectable. An ELISA- based detection of NP-specific lgM antibodies is usually performed for laboratory diagnosis of an acute hantavirus infection[15].

Serological tests for the detection of lgM and lgG antibodies based on indirect immune fluorescence assays, strip immunoblot, or enzyme-liked-immunno sorbent assays are diagnosis procedure[16].

Treatment

At present there are no antiviral drugs that are applicable to cure hantavirus infections. Ribavinrin (1,4,3-D-ribofuranosy 1-1,2-4-trazole-3-carboxamide), a guanosine-analog, was shown to posses anti-hantavirus activity [17].

Ribavinrine an antiviral drug may be of some benefit. But what appears to help most is being hospitalized early, monitored carefully, and treated with life-sustaining fluids medications that help normalize heart rate and breathing [18].

Prevention

Because infection with some hantavirus species results in high morbidity and mortality rates keeping homes and the near surrounding area rodent-free, for example, by eliminating crawl spaces and debris and removing food sources to make homes and work areas unattractive for rodents. To develop an effective and safe vaccine against hantaviruses applying vaccination techniques varying from killed virus to recombinant DNA technology [19].

Since infection is thought to occur by inhalation of rodent wastes (excreta), prevention is aimed toward eradication of rodents in houses and avoidance of exposure to rodent excretes in rural settings.

HANTAVIRUS IN KIDNEY DISORDER

Hantavirus hemorrhagic fever with renal syndrome (HFRS) is a group of clinically similar illnesses caused by species of hanta viruses from the family Bunyaviridae. It is also known as Korean hemorrhagic fever, epidemic hemorrhagic fever, and nephropathies epidemica [20].

The course of the illness can be split into five phases

- Febrile phase: Symptoms include redness of cheeks and nose, fever, chills, sweaty palms, diarrhea, malaise, headaches, nausea, abdominal and back pain, respiratory problems such as the ones common in the influenza virus, as well as gastro-intestinal problems. These symptoms normally occur for three to seven days and arise about two to three weeks after exposure.
- Hypotensive phase: This occurs when the blood platelet levels drop and symptoms can lead to tachycardia and hypoxemia. This phase can last for 2 days.
- Oliguric phase: This phase lasts for three to seven days and is characterized by the onset of renal failure and proteinuria.
- Diuretic phase: This is characterized by diuresis of three to six liters per day, which can last for a couple of days to weeks.
- Convalescent phase: This is normally when recovery occurs and symptoms begin to improve [21].

This syndrome can also be fatal. In some case, it has been know to cause permanent renal failure.

There is no cure or vaccine for HFRS. Treatment involves supportive therapy including renal dialysis. Treatment with ribavirin in China and Korea, administered within 7 days of onset of fever, resulted in a reduced mortality [22].

CONCLUSION

The increased awareness of hanta virus infection should be given by medical practioner early in the diagnosis and prevention of infection by keeping homes and near surroundings area rodent – free, developing effective and safe vaccine are the ways to decrease the emergence of hantavirus infection.

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CONFLICT OF INTEREST

We declare that we have no conflict of interest.

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