

Social Implications of the Yosemite Hantavirus Outbreak

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Abstract

In 2012, Hantavirus-an RNA virus that is commonly carried by mice-impacted overnight tourists who camped at Yosemite National Park. I will be exploring the particular cultural and historical setting of Hantavirus, specifically Sin Nombre virus. Literature regarding the Yosemite Hantavirus outbreak will answer two essential questions: 1) What are the short-term societal impacts of the Yosemite outbreak? 2) What are the long-term societal impacts of the Yosemite outbreak? These two guestions will be analyzed based on the contexts of local versus global impacts and positive versus negative impacts. By understanding the impacts of Hantavirus on the surrounding community, I hope to highlight the strengths and weaknesses of current and past response protocols and encourage more effective preparation for future Hantavirus outbreaks.

Introduction and Background

Hantavirus causes two specific health complications: Hantavirus Pulmonary Syndrome (HPS) and Hemorrhagic Fever with Renal Syndrome (HFRS) ("Hantavirus", 2016). HPS, commonly caused by the Sin Nombre virus, directly impacts the respiratory system causing early flu-like symptoms including fever, muscle aches, headaches, vomiting, nausea, diarrhea, dizziness, and chills. Late symptoms-approximately four to five days post early symptoms of HPS include fluid build-up in the lungs and dyspnea (shortness of breath). HPS is commonly found in the New World (including North and South America) Hantavirus. Deer mice and rats are reservoirs for Hantavirus in the New World, and humans can contract the virus by coming into contact with or breathing in virus from infected rodent urine, feces, and saliva. Human to human transmission of Hantavirus has not been reported (Safronetz et al., 2016, p. 7114-7119). HFRS is a form of Hantavirus commonly found in the Old World—Asia and Europe—and includes Seoul virus carried by Rattus rattus, Puumala virus

carried by Myodes glareolus, Dobrava virus carried by Apodemus flavicollis, and Saaremaa virus carried by Apodemus agrarius. Similar to the Sin Nombre virus, the Old World Hantavirus can be transmitted to humans by exposure or contact with rodent urine, feces, or bodily fluids. Symptoms of HFRS also include nausea, fever, chills, abdominal pain, and blurred vision. However, the late symptoms of HFRS are more severe than those of HPS and possess a 5-15% fatality rate for patients in the United States. Late symptoms include vascular leakage, severe hypotension, and acute kidney failure ("Hemorrhagic Fever with Renal Syndrome HFRS", 2008, p. 508-509).

HPS and HFRS are often misdiagnosed during the initial stages due to similarities with the common cold and influenza. However, patients who state previous or potential exposure to rodent infested environments complete serologic testing for Hantavirus antigen presence via immunohistochemical staining. Specific diagnostic measures for HFRS include thrombocytopenia (low number of platelets), urine tests for high levels of albumin, and urine tests for blood presence. ELISA, immunofluorescence staining, and thrombocytopenia are used as diagnostic measures for HPS (MacNeil, 2011, p. 238-140). No antiviral drugs or vaccinations are available for Hantavirus; therefore, treatments are catered towards regulating symptoms (Lee, 1996, p. 253-257). Early detection of Hantavirus will allow for more effective treatment of the symptoms including careful fluid and electrolyte monitoring for HFRS and oxygen therapy for severe cases of respiratory distress for HPS (Cosgriff, 1991, p. 97-98). The pathophysiology of Hantavirus impacts the epidemiological response towards HPS and HFRS, particularly the Yosemite HPS outbreak.

Societal History of Hantavirus Internationally

HFRS first shed light on the international impacts of Hantavirus, when approximately 3,000 cases of the disease were reported in South Korea in 1951, a time when UN forces were fighting on the 38th parallel during the Korean War. HFRS had most likely been present in Eurasia, specifically Korea, Manchuria, and Russia, for centuries with the first records found in 1913; a milder account of Puumala fever was discovered in Finland in the early 1930s (Cameron, 2011, p. 1289-1290). Hantaan virus (HTNV) surveillance demonstrated the presence of viruses similar to that of HTNV in Far East Asia, China, and South Korea in the following species of rodents: Apodemus agrarius and A. peninsulae. In Europe, the Dobrava virus was found in the following species of rodents: Apodemus flavicollis, A. agrarius, and A. ponticus.

Recorded cases of Asian urban HFRS in the 1980s are linked to the Seoul virus in Asian, and recorded cases of European urban nephropathia epidemic (a milder version of HFRS) in the 1930s are linked to the Puumala virus (Jonsson, 2010, p. 420-421). With the greater availability of the Hantavirus antigen, urban rodents (Rattv. norvegicus and Rattus rattus) have been found to harbor the virus after transmission from humans. This urbanized version of HFRS has now been serologically identified in North and South America, New Guinea, India, the pacific islands (including the Philippines, Hawaii, Taiwan, and Fiji), and Africa. Isolated forms of the Hantavirus have been reported to be in use for medical research in Japan, Belgium, France, United Kingdom, and Korea. Currently, HFRS remains an international public health threat as approximately 150,000-200,000 patients are hospitalized due to Hantavirus (Lee, 1996, p. 260-265). With the globalization of medicine through advanced transportation and communication, both the Hantavirus disease and people's awareness of the disease have spread across the world.

Societal History of Sin Nombre Virus in the United States

Previous cases of HFRS in the United States were reported around 1862 and 1863—during the Civil War era; however, the lack of serological testing prevented confirmation of Hantavirus. The first recorded outbreak of Hantavirus was in 1993 in the Four Corners (Arizona, Colorado, New Mexico, and Utah). The Hantavirus in the Four Corners was characterized by the HPS causing virus Sin Nombre. Serological testing of Sin Nombre represented a cross reactivity between the Old World Hantavirus (HFRS) and human sera (Jonsson, 2010, p. 437-439). The first reported patient of the 1993 outbreak was a previously physically fit and healthy Navajo man from New Mexico, who experienced shortness of breath and died shortly afterwards, and his fiancé had died several days before, demonstrating similar symptoms of respiratory distress. Dr. Bruce Tempest of the Indian Health Service then identified five additional previously healthy patients who died of acute respiratory distress. After serological testing failed to identify the cause of death, the following departments were immediately notified: Center for Disease (CDC), the Indian Health Service, the University of New Mexico, the Navajo nation, as well as the state health departments of New Mexico, Colorado, and Utah. In order to determine the natural reservoirs for the unknown virus, rodents found near or inside the living areas of the patients were trapped and used for tissue testing. After approximately 1,700 rodent trappings, the virus was finally isolated in November 1993 and identified as the Sin Nombre virus (Khan, 1997, p. 1297-3000). The surge of the HPS outbreak in 1993 is attributed to a drastic increase in the rodent population after a long drought period. The high number of mice increased the chances of human-rodent exposure.

In an analysis of the first one hundred patients in the United States infected with HPS since 1993, Hantavirus outbreaks were common during the spring and early-summer timeline. There was not a significant gender difference; 54% of patients were males. The ethnic distribution of the first one hundred patients was 63% Caucasian, 35% Native American, and 2% African American, and the average age for the cases was 34.9 years (Ksiazek, 1995, p. 121-124). Through tight regulation and early diagnosis measures, the Hantavirus outbreak is being monitored and regulated from reaching pandemic levels; however, Hantavirus HPS continued to impact people in the United States. From 1993 through 2011, HPS has impacted 587 people in the United States. In the summer of 2012, Hantavirus reemerged, attacking tourists visiting Yosemite National Park.

Short-Term Societal Impacts of the Yosemite Hantavirus

Initially, ten tourists from three different states who stayed overnight at Yosemite National Park were experiencing respiratory complications, characteristic of HPS symptoms. For the first eight patients who were identified, five required ventilator assistance in the intensive care unit and the other three patients died. After surveying the patients, nine out of ten tourists resided in the Curry Village Yosemite signature tent cabins overnight, which were marked by rodent nests and tunnels in the foam insulation of the walls. 50% of tourists who stayed in signature tents were from California, 30% from other states, and 20% from other countries. For epidemiological analysis, 73 deer mice were trapped and completed serological testing, and 14% of the trapped mice tested positive for Sin Nombre virus. Patients who were infected with HPS engaged in similar activities as tourists not impacted by the disease. As an immediate short-term response to the Yosemite outbreak, the park shut down and dismantled all 91 signature tents to prevent further transmission. 1,300 buildings were inspected, rodent exclusion practices were performed when necessary, and rodent population surveillance was put into place via various trappings throughout the park. (Hartline, 2013, p. 978-982). Addressing this outbreak incentivized the collaboration of the CDC, California Department of Public Health (CDPH), and the National Park Service (NPS) Office of Public Health.

From August 27th to September 17th, 2012, the park presented every tourists with informational handouts regarding Hantavirus, posted educational messages in common areas, and trained park employees about preventative measures. The park also contacted approximately 10,000 tourists who had stayed overnight in the signature cabins and encouraged them to immediately seek medical attention if they presented any symptoms of HPS. The Yosemite outbreak was different from that of the 1993 outbreak due to the guick spread of the disease but also quicker identification and response to the disease. The patients from the Yosemite outbreak were also exposed to a smaller geographic region than the patients from the four corners (Nunez, 2014, p. 386-393). However, the previous response tactics that were implemented during the 1993 outbreak aided the efforts to tackle the Yosemite outbreak.

Because 20% of tourists were from countries outside of the United States, the park officials meticulously analyzed paperwork for all international visitors and contacted public health officials for 39 countries. One specific example includes the UK's response towards the Yosemite outbreak. The UK Health Protection Agency used information from the US National Park Service to contact approximately one hundred travellers deemed to have a high risk of being exposed to Sin Nombre. This response allowed for public media coverage and publications in academia (e.g. Vanya Gant's paper regarding the Yosemite outbreak in the New England Journal of Medicine), furthering awareness of the disease and its symptoms (Roehr, 2012). This collaborative response on an international scale exemplified the advantages of quick acknowledgment and notification of zoonotic outbreaks.

Long-Term Societal Impacts of the Yosemite Hantavirus

The Hantavirus outbreak only temporarily impacted the tourism industry for Yosemite National Park. The quick response and additional precautionary measures encouraged more tourism after the successful suppression of HPS. Currently, tourism in Yosemite National Park generates over \$378 million each year and involves 5,162 jobs for the local community ("Economics", 2015). The Yosemite outbreak lead to the restructuring of preventative measures for park employees. The program emphasizes extensive use of cleaning practices, use of personal protective equipment (PPE), and education of Hantavirus safety training and knowledge of HPS (Wilken, 2015, p. 663-664). The high fatality and quick spread of the Yosemite HPS outbreak prompted the international medical community to conduct further research on the development of a Hantavirus vaccination.

Hantavax, a Korean Hantaan virus vaccine, demonstrated high antibody titers and neutralizing antibodies over the course of a twelve-month vaccination period. No serious adverse effects were reported; therefore, Hantavax is a potential vaccination that may be introduced by the World Health Organization after further testing. Pharmaceutical companies were reluctant to fund Hantavirus vaccinations due to the low instances of Hantaan infections in the majority of the world, especially since Hantavirus has been reported in countries wellequipped with medical resources to aid patients without the need for antiviral drugs or vaccinations; however, the Yosemite outbreak once again reminded the medical community of the advantages of a Hantavirus vaccination as a crucial preventative measure (Maes, 2009, p. 68-73). Because Hantavirus is indiscriminate and can impact people regardless of socioeconomic standards, gender, or ethnicity, the Yosemite outbreak was an important stimulus for raising awareness regarding its global impacts.

Conclusion

Hantavirus has existed in society for centuries as HPS and HFRS, impacting patients from Asia to the United States. As the medical community conducts further research regarding HPS and HFRS, the virus continues to adapt to the changing environment. The 2012 Yosemite outbreak served as a reminder that effective public health responses, communication between various healthcare departments, and development of novel preventative measures regarding Hantavirus are necessary in combatting this ever-changing disease.

References

Cameron, J. S. (2001). The history of viral haemorrhagic fever with renal disease (hantavirus). Nephrology Dialysis Transplantation, 16(6), 1289-1290. doi:10.1093/ndt/16.6.1289

Cosgriff, T. M. (1991). Mechanisms of Disease in Hantavirus Infection: Pathophysiology of Hemorrhagic Fever with Renal Syndrome. Clinical Infectious Diseases, 13(1), 97-107. doi:10.1093/clinids/13.1.97

Economics. (2015, April 21). Retrieved May 20, 2016, from http://www.nature. nps.gov/socialscience/economics.cfm

Hantavirus. (2016, January 12). Retrieved May 20, 2016, from http://www.cdc. gov/hantavirus/

Hartline, J., Mierek, C., Knutson, T., & Kang, C. (2013). Hantavirus infection in North America: A clinical review. The American Journal of Emergency Medicine, 31(6), 978-982. doi:10.1016/j.ajem.2013.02.001

Hemorrhagic Fever with Renal Syndrome (HFRS). (n.d.). Encyclopedia of Public Health, 672-672. doi:10.1007/978-1-4020-5614-7_1516

Jonsson, C. B., Figueiredo, L. T., & Vapalahti, O. (2010). A Global Perspective on Hantavirus Ecology, Epidemiology, and Disease. Clinical Microbiology Reviews, 23(2), 412-441. doi:10.1128/cmr.00062-09

Khan, A. S., Khabbaz, R. F., Armstrong, L. R., & Holman, R. C. (1997). Hantavirus pulmonary syndrome: The first 100 US caases. The Journal of Infectious Diseases, 173(6), 1297-1303. doi:10.1093/infdis/173.6.1297

Ksiazek, T. G., Peters, C. J., Rollin, P. E., Zaki, S., & Nichol, S. (1995). Identification

of a new North American hantavirus that causes acute pulmonary insufficiency. American Journal of Tropical Medicine and Hygiene, 52(2), 117-123. Retrieved May 20, 2016, from http://europepmc.org/abstract/med/7872437

Lee, H. W. (1996). Epidemiology and Pathogenesis of Hemorrhagic Fever with Renal Syndrome. The Bunyaviridae, 253-267. doi:10.1007/978-1-4899-1364-7_10

MacNeil, A., Nichol, S. T., & Spiropoulou, C. F. (2011). Hantavirus pulmonary syn drome. Virus Research, 162(1-2), 138-147. doi:10.1016/j.virusres.2011.09.017

Maes, P., Clement, J., & Ranst, M. V. (2009). Recent approaches in hanta virus vaccine development. Expert Review of Vaccines, 8(1), 67-76. doi:10.1586/14760584.8.1.67

Núñez, J. J., Fritz, C. L., Knust, B., Buttke, D., Enge, B., Novak, M. G., . . . Vugia, D. J. (2014). Hantavirus Infections among Overnight Visitors to Yosemite National Park, California, USA, 2012. Emerg. Infect. Dis. Emerging Infectious Diseases, 20(3), 386-393. doi:10.3201/eid2003.131581

Roehr, B. (2012). US officials warn 39 countries about risk of hantavirus among travellers to Yosemite. Bmj, 345(Sep10 1). doi:10.1136/bmj.e6054

Safronetz, D., Prescott, J., Feldmann, F., Haddock, E., Rosenke, R., Okumura, A., . . . Feldmann, H. (2014). Pathophysiology of hantavirus pulmonary syndrome in rhesus macaques. Proceedings of the National Academy of Sciences, 111(19), 7114-7119. doi:10.1073/pnas.1401998111

Wilken, J. A., Jackson, R., Materna, B. L., Windham, G. C., Enge, B., Messenger, S., . . . Roisman, R. (2015). Assessing prevention measures and sin nombre hantavirus seroprevalence among workers at Yosemite National Park. Am. J. Ind. Med. American Journal of Industrial Medicine, 58(6), 658-667. doi:10.1002/ajim.22445