G97 Hantavirus Pulmonary Syndrome: A Case Report

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After this presentation, attendees will be able to describe the geographical distribution of Hantavirus, its host, routes transmission,detail the pathogenesis and symptomatology infection, and characterize the Hantavirus Pulmonary Syndrome (HPS), and describe expected autopsy findings

This presentation will impact the forensic science community by familiarizing its audience with the features of the HPS, its clinical presentation, autopsy findings, and pertinent postmortem ancillary testing

Hantaviruses include a related group of RNA viruses of the Bunyaviridae family. It is transmitted by rodent hosts, mainly via aerosolized feces and urine, causing only incidental disease in humans. HPS, however rare, is a serious life-threateninig condition that needs early diagnosis and support therapy, since symptoms can develop very rapidly (within 24 hours).

Presented is the case of a 20-year-old woman with no significant past medical history who complained of having headache, cough, muscle and stomach aches, fever, and shortness of breath for several days. She was seen at a clinic and treated as an outpatient with ciprofloxacin. Two days later, she was taken to an urgent care facility due to acute onset of shortness of breath. She was admitted to the Intensive Care Unit (ICU) and died shortly after her admission. The admission Computed Tomography (CT) scan showed extensive bilateral pleural effusions.

Autopsy revealed marked lung edema and large bilateral straw-colored pleural effusions (right, 850mL; left, 550mL). Microscopically, the lungs showed extensive areas of intra-alveolar fluid, with fibrin depositiona and focal hyaline membrane formation. Anterstitial mononuclear cell infiltrate with immunoblasts was identified in the lungs, liver, and spleen. A serum assay was positive for *Sin Nobre* Hantavirus antibodies.

While the incidence of Hantavirus infections has been relatively low (551 cases reported in the United States between 1993 and 2011), the mortality rate is alarmingly high (about 35%). This is due to a virus-induced vigorous immune response within vascular epithelium leading to vascular instability, especially within the lungs. Capillary dilation and leakage lead to the more severe manifestations of disease: shock and respiratory failure.

Without an intermediate host, each rodent host serves as both the primary host and reservoir in nature, transmitted via saliva, respiratory tract secretions, feces, and urine, causing chronic infection in rodents and only incidental disease in humans, generally by inhalation of aerosols of these body fluids or of virus-laden dust. Other means of infection include bites from infected animals, contamination of cutaneous wounds or mucous membranes, or ingestion of contaminated food. With the exception of the Andes virus of Argentina, the virus is not transmitted between humans.

At particular risk are individuals living or working in small, dark spaces with poor ventilation and known rodent presence, perhaps following a period of environmental conditions favorable to rodent reproduction. A classic case might describe exposure following cleaning of a shed, vacation home, or livestock feed container.

The natural history of disease of HPS generally includes three phases that follow an incubation period that lasts from two to four weeks: prodrome—characterized by sudden onset of nonspecific fever, malaise, myalgia, headache, chills, nausea, or diarrhea, and lasting about three to five days; cardiopulmonary—with nonproductive cough and varying degrees of shortness of breath, with associated tachypnea and mild hypotension and, laboratory evaluation—will reveal hemoconcentration (elevated hemoglobin/hematocrit), thrombocytopenia, neutrophilic leukocytosis with left shift, and reactive lymphocytes. At this stage, the patient is at the most risk for massive fluid shifts and pulmonary edema. The patients who die usually succumb to progressive cardiac insufficiency. If this phase is contained, the convalescence—or diuretic phase—begins, with usually little significant long-term sequela, though exertional dyspnea may persist for up to three months.

Considering the speed with which Hantavirus can overwhelm its victims, rapid diagnosis is essential in the clinical setting. In the postmortem setting, this is no less true, as a quickly recognized and diagnosed Hantavirus infection can allow medical investigators to identify family or coworkers at risk of a similar decline.

Serology remains the most common means of Hantavirus diagnosis, with IgM-capture ELISA providing a presumptive diagnosis. This can be performed in the postmortem setting, but the authors recommend making every effort to get an antemortem sample, since serum extraction in postmortem samples can become difficult. Detection of hantaviral antigen in tissue biopsy or other autopsy specimens can also be pursued. Other diagnostic modalities include viral RT-PCR from blood or fixed tissue or immunohistochemical demonstration of viral antigen in fixed tissue.

Hantavirus, *Sin Nombre* Virus, Pulmonary Syndrome

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