Case Report

Hantavirus Cardiopulmonary syndrome: Another Reason to Avoid Mice

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Mark Kennedy, MD[1], Omar Mctabi, DO[1], and Christopher Rickman, MD[1]

Abstract

Hantavirus cardiopulmonary syndrome is a severe illness transmitted by rodent excretions. We describe a case of a 24-year-old man who presented to the emergency department with cough, shortness of breath, chills, myalgias, nausea, and diarrhea. Physical examination and laboratory analysis revealed signs of respiratory distress and thrombocytopenia. The trajectory of his illness led to acute respiratory distress syndrome (ARDS) and hemodynamic instability. Serum testing was positive for hantavirus IgM and IgG antibodies. The patient was managed with supportive care and improved. This case highlights the importance of considering hantavirus when managing patients who develop thrombocytopenia, ARDS, and hemodynamic instability in the appropriate clinical setting.

Keywords

hantavirus, cardiopulmonary syndrome, thrombocytopenia

Introduction

Hantavirus is a rare cause of zoonotic disease transmitted by inhaled, aerosolized excretions from infected rodents.[1] Of the 2 clinical syndromes associated with the virus, hantavirus cardiopulmonary syndrome (HCPS) is an emerging disease with a mortality of 40%.[1,2] Its presence in the United States was first identified in 1993.[3] Since surveillance for the disease began, 821 cases of HCPS have been reported to the Center for Disease Control and Prevention (CDC) from 1993 to 2021.[4] The clinical course mimics flu-like illnesses with thrombocytopenia, followed by a rapid progression to hemodynamic instability and respiratory failure.[1,2] We describe a case of a young man who was found to have HCPS.

Case Presentation

A 24-year-old man with no notable past medical history presented to our hospital for cough, shortness of breath, chills, myalgias, nausea, and diarrhea. His symptoms began 7 days ago with upper respiratory symptoms including a productive cough of yellow sputum and shortness of breath on exertion. Approximately 4 days later, he developed myalgias, nausea, and watery diarrhea, which prompted him to present to the emergency department. He was diagnosed with community-acquired pneumonia and discharged from the emergency room with doxycycline. Despite taking 2 doses of doxycycline at home, he developed a rapidly progressing dyspnea at rest, for which he presented to our emergency department again the following day.

On physical examination, the patient was afebrile at 37.4°C with a blood pressure of 122/81 mm Hg. He presented with tachypnea and tachycardia with a respiratory rate of 30 breaths per minute and a heart rate of 132 beats per minute. Oxygen saturation was 87% on room air. He appeared diaphoretic with increased work of breathing and accessory muscle use. Pulmonary auscultation revealed bilateral rales over the upper and lower posterior lung fields. Laboratory analysis showed an elevation in white blood cells to 18,000 per mm^3, with 68% neutrophils and 6% band neutrophils. Platelets were 31,000 per mm^3, sodium was 129 mmol/L, and lactic acid level was 3.94 mmol/L. Arterial pH was 7.43, the partial pressure of carbon dioxide was 31.2 mm Hg, and the partial pressure of oxygen was 83.5 mm Hg. Multiplexed nucleic acid testing for common respiratory viruses, including influenza A and B, Respiratory Syncytial Virus, coronaviruses, and parainfluenza virus, was negative. The patient denied a recent history of travel and reported his occupation

[1]Community Hospital, Grand Junction, CO, USA

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Corresponding Author:
Mark Kennedy, 657 Trinity Way Unit A, Grand Junction, CO 81505, USA.
Email: Mark.al.kennedy@gmail.com

as a handyman, having recently constructed an outdoor patio in Western Colorado.

Computed tomography with angiography of the chest demonstrated patchy areas of ground glass opacities bilaterally at the lower lobes in addition to prominent interlobular septa, small bilateral pleural effusions, and reflux of contrast at the inferior vena cava, suggesting right heart dysfunction.

Levofloxacin was administered in addition to intravenous fluids for the treatment of community-acquired pneumonia and sepsis. His recent visit to the emergency department, failure to improve with doxycycline, and severe presentation of pneumonia were concerning for pseudomonas infection, for which a respiratory fluoroquinolone was initially chosen. Legionella was also considered due to hyponatremia, although a macrolide in addition to a beta-lactam antibiotic would have sufficed as initial therapy if pseudomonas was not also being considered.

Over the next 6 hours, the patient's oxygen requirements increased from 5 L by nasal cannula to high flow nasal cannula with a fraction of inspired oxygen (FiO2) of 75% at 15 L per minute of flow. Despite escalating oxygen delivery, the patient became progressively lethargic with increased use of accessory muscles for respiration. Chest X-ray showed worsening bilateral interstitial opacities. He was intubated 16 hours following admission. Subsequent arterial blood gas assessment revealed a PaO_2/FiO_2 (P/F) ratio of 86, consistent with severe acute respiratory distress syndrome (ARDS). Following intubation, mean arterial pressure dropped below 65 mm Hg, for which continuous norepinephrine was initiated. Subsequent management included providing the patient with a protective lung strategy on mechanical ventilation and escalation of antibiotics to vancomycin and piperacillin/tazobactam. Methylprednisolone was also administered for the treatment of severe community-acquired pneumonia.

On the second day of hospitalization, blood pressure was 95/63, temperature was 37.9°C, pulse 96 beats per minute, respiratory rate 25 breaths per minute, and SpO_2 of 94%. He was on volume control ventilation with a respiratory rate of 22 breaths per minute, tidal volume of 400 mL (which is 6 m/kg of his ideal body weight), positive end-expiratory pressure (PEEP) of 10 mm Hg, and FiO_2 of 100%. Additional laboratory analysis revealed negative legionella and streptococcus pneumoniae urine antigens, negative HIV 1 and 2 antibodies, blood cultures without growth, and urine drug screen positive for tetrahydrocannabinol. Transthoracic echocardiogram was normal other than a small anterior pericardial effusion.

Additional history was obtained from the patient's mother following intubation, who mentioned the patient had a pet guinea pig and other rodents living in his residence. This led to a suspicion of HCPS, for which serum testing of hantavirus antibodies to IgM and IgG was obtained. He was discharged without requiring supplemental oxygen following recovery. Hantavirus antibodies to IgM and IgG resulted in positive following discharge.

Discussion

Hantaviruses are negative-sense, single-stranded RNA viruses associated with the geographic distribution of rodents.[1] Sin Nombre Virus is the most common cause of HCPS in the United States and is transmitted by deer mice (*Peromyscus maniculatus*) found throughout North America.[5,6] Our patient was likely exposed to the virus via rodent excretions from occupational exposure or pet rodents while living in Western Colorado, where the majority of Hantavirus cases have been reported to the CDC.[3]

The clinical course begins as a 3- to 5-day prodrome of myalgias, fever, nausea, and vomiting.[7] This short, flu-like illness is later accompanied by a rapid progression to the cardiopulmonary phase, distinguished by cardiogenic shock and respiratory failure requiring mechanical ventilation.[2] Those who improve enter a stage of convalescence with complete recovery.[1] During the cardiopulmonary phase, a presumptive diagnosis can be made if the following 4 out of 5 criteria are met: thrombocytopenia, left shift in the granulocytic lineage, absence of toxic granulation in the myeloid series, hemoconcentration, and an immunoblast population greater than 10% of the total leukocyte count.[8] Our patient met 3 of these 5 criteria.

A diagnosis is confirmed with polymerase chain reaction and enzyme-linked immunosorbent assay to detect IgM and IgG antibodies directed against hantavirus nucleocapsid proteins.[1,8]

Once a diagnosis of HCPS is confirmed, patients should be transferred to a health care facility with access to extracorporeal membrane oxygenation (ECMO) and critical care units.[8] Currently, there is no specific therapy available for the treatment of HCPS.[5,9] Vasopressors, mechanical ventilation, and ECMO may be required.[5] ECMO should be considered over mechanical ventilation, as this has been shown to improve mortality outcomes.[5,8] Our patient's respiratory failure and circulatory shock were managed with mechanical ventilation and norepinephrine alone due to a delay in obtaining IgM and IgG antibody results from a send out laboratory blood sample. The role of steroids remains controversial in the treatment of HCPS, but had not demonstrated an increase in serious adverse events in 1 controlled trial.[10] The use of corticosteroids in our patient was initially administered in an attempt to attenuate the immune response leading to ARDS and new evidence demonstrating corticosteroids reduce 28-day mortality in cases of severe community-acquired pneumonia.[11] However, additional controlled trials with a greater number of participants are needed to support or reject its clinical utility in managing HCPS.

Conclusion

Hantavirus cardiopulmonary syndrome is a viral infection most commonly caused by the Sin Nombre Virus in the United States.[5] It is transmitted by inhaling the excrement of infected rodent excretions.[1] Recognition can easily go undetected due to its similar presentation to other illnesses with

flu-like symptoms and thrombocytopenia. The disease course results in severe respiratory and circulatory failure, resulting in a 40% mortality.[1] Diagnosis is confirmed with antibodies to IgM and IgG in addition to polymerase chain reaction.[8] Treatment is supportive, with ionotropic medications, mechanical ventilation, and extracorporeal membrane oxygenation.[5]

Author contributions

M.K., MD, contributed to drafting the manuscript and conducting the literature review. O.M. DO, contributed to the revision of the manuscript and has approved of its content. C.R., MD, contributed to the revision of the manuscript and approved of its content.

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Informed Consent

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ORCID iD

Mark Kennedy https://orcid.org/0009-0005-9377-0980

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