Hantavirus Pulmonary Syndrome Causing Non-Cardiogenic Pulmonary Edema

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INTRODUCTION
Hantavirus pulmonary syndrome (HPS) consists of an incubation period followed by a flu-like prodrome, progressing to a cardiopulmonary phase of variable severity. We present a case of Hantavirus pulmonary syndrome causing non-cardiogenic pulmonary edema.

LEARNING OBJECTIVES
1. Recognizing a presentation of hantavirus pulmonary syndrome
2. Identifying clinical hallmarks of hantavirus pulmonary syndrome in the U.S.

CASE
Presentation
A 52 year-old man from northern California with a history of hypertension went on a 3-week summer vacation, first to Yosemite, then to Niagara Falls and finally to Yellowstone. Two nights before the end of the trip he developed nausea. Over the next two days he developed fevers, myalgias, abdominal pain and a headache, prompting him to present to the emergency room. He had an unremarkable workup other than mild thrombocytopenia with platelets of 111,000. He was treated with Tamiflu but continued to be symptomatic. Two days later he developed a cough and dyspnea. He visited his primary care provider and was treated for community acquired pneumonia with doxycycline. Later that day he became acutely dyspneic, complaining that he had "water in his lungs" and returned to the emergency room. He rapidly developed hypoxic respiratory failure and was intubated and started on vasopressors.

Exam
Fever to 103.1°F, HR 122, MAP 50, RR 30-40s, oxygen saturation <90% on 10L non-rebreather mask.
Chinese male, alert and responsive, in acute respiratory distress with sternocleidomastoid retractions, frequent cough and diffuse rhonchi. Alert and responsive. Normal heart exam, no JVD. No edema, rashes or bites.

Laboratory Studies
Hgb16.8/U Hct 47.5, Platelets 73K–54K, bands 38%, no toxic granulation, peripheral blood smear: see figures 3 and 4.
Na 127, creatinine 1.5, AST 86, ALT 98, LDH 489, lactate 3.2, albumin 2.7, PTT 43.8.
Chest x-ray: See figures 1 and 2. TTE: normal systolic function

Clinical Course
Pax02/Flav was <100 mm/kg despite PEEP > 5 cm H2O, consistent with severe ARDS. He was started on empiric broad-spectrum antibiotics with ventilator management according to the ARDSNet protocol. HD 2 his AKI resolved, norepinephrine was discontinued and he was aggressively diuresed. HD 4 his fevers subsided and antibodies returned positive for the hantavirus Sin Nombre virus. HD 6 he was extubated.

DISCUSSION
• Hantaviruses are RNA viruses in the family Bunyaviridae. "Old World" hantaviruses in Europe and Asia are associated with hemorrhagic fever and renal syndrome (HFRS). "New World" hantaviruses in North, Central and South America cause hantavirus pulmonary syndrome (HPS).
• HPS is a febrile illness characterized by bilateral diffuse interstitial edema, with respiratory compromise, and often occurring in a previously healthy person.
• The clinical course is variable, with an incubation period of up to 6 weeks followed by a viral prodrome. This may be followed by a cardiopulmonary phase with abrupt onset of cough and dyspnea, rapidly progressing to pulmonary edema.
• Several hantavirus species cause HPS. Each is associated with a specific rodent carrier. Most cases in the U.S. are attributed to the Sin Nombre virus. Transmission to humans typically occurs via inhalation of airborne virus particles excreted by deer mice (carrier of SNV).
• The exact mechanism of pathogenesis is unclear but is proposed to be via combination of direct effects on endothelial cells and indirect immunogenic effects causing pulmonary endothelial leakage.
• Differential diagnosis in an immunocompetent host includes: atypical pneumonia (Chlamydia, Mycoplasma, Legionella), pneumonic plague, sepsis/ARDS, Influenza, SARS, Coccidiomycosis, acute interstitial pneumonia, collagen vascular disease.
• Diagnosis of HPS: + IgM or fourfold rise in IgG antibodies to hantavirus; IgG or IgM antibodies to specific hantiviruses
• Presumptive diagnosis may be made with 4/5 of the following hematologic findings: thrombocytopenia, hemococoncentration, left shift, lack of significant toxic granulation in neutrophils, more than 10% of lymphocytes with antibodies to specific hantavirus
• HPS should be suspected in otherwise healthy patients with a typical prodrome, known exposure risk and thrombocytopenia. Early recognition of HPS may increase the likelihood of survival.

REFERENCES