Hantavirus Pulmonary Syndrome: High-resolution Computed Tomography Findings☆

Síndrome pulmonar por Hantavirus*: hallazgos en la tomografía axial computarizada de alta resolución*

To the Editor,

We retrospectively reviewed the records of 8 adult patients with confirmed hantavirus pulmonary syndrome (HPS), aiming at describing high-resolution computed tomography (HRCT) findings. The patients were examined between 2003 and 2014 in 6 tertiary hospitals in Brazil. Diagnoses of HPS were based on medical histories, clinical course, and imaging findings. Serological tests (ELISAs) were positive for Hantavirus in all patients. Although the HRCT findings of Old World hantaviruses have been well described, to our knowledge no study has examined HRCT findings in a series of patients with HPS. In our study, the main HRCT findings were ground glass opacities (GGOs) and smooth interlobular septal thickening, observed in all patients (Fig. 1). However, the crazy-paving pattern was observed in only 3 cases. Pleural effusion and peribronchovascular thickening were observed in 5 cases. Four patients had small nodules, and only 1 had foci of consolidation. Abnormalities were bilateral and diffuse in all patients.

HPS is an emerging zoonotic disease caused by hantavirus, a spherical and enveloped single-stranded RNA virus belonging to the family Bunyaviridae. In the Americas, natural reservoirs of the virus are rodents of the subfamily Sigmodontinae.[1,2] The genus Hantavirus consists of several viruses classified into 2 groups, each associated with a different clinical syndrome: Old World hantaviruses, which cause hemorrhagic fever with renal syndrome or nephropathia epidemica; and New World hantaviruses, related to HPS.[1,3] HPS was first recognized as a clinical entity in the United States in 1993. On average, approximately 200 cases of HPS per year are reported in the Americas, and although the number of cases is much smaller than that of HFRS, its average case fatality is about 40%.[4]

Both syndromes primarily involve young adult males, and transmission is related to occupational activities, such as veterinary medicine, farming, and related professions.[1] Hantavirus can affect humans after inhalation of aerosolized virus particles from urine, saliva, or dried excreta of reservoir rodents. Person-to-person transmission has been reported in a few cases, generally associated with a specific strain, the Andes virus.[5]

The clinical presentation of HPS is usually nonspecific, and in most cases presents as flulike symptoms. A cough (initially nonproductive) typically signals the transition to the cardiopulmonary phase, in which a fulminant capillary leak syndrome may lead to rapidly progressive pulmonary edema and shock.[5] Clinical manifestations develop after an incubation period of about 2–3 weeks, and usually comprise 3 well-demarcated phases. The prodromal phase is characterized by nonspecific, flulike manifestations that last from 2 to 5 days. The cardiopulmonary phase typically starts with a dry cough that rapidly becomes productive, with mucus and bloody sputum, along with respiratory failure and cardiovascular shock. Laboratory findings during this phase include marked leukocytosis with a leftward shift, thrombocytopenia, elevated serum lactate dehydrogenase and aspartate aminotransferase levels, hemoconcentration, and low serum albumin concentration due to capillary leakage.[1–3,5] Improvements in oxygenation, diuresis, and hemodynamic stabilization are the hallmarks of the convalescent phase. Although a presumptive diagnosis of HPS can be made based on the patient's clinical history and radiologic findings, confirmation of the diagnosis requires virus-specific diagnostic tests, such as serological tests (ELISA), reverse transcription, and/or PCR. ELISA results for all patients in our sample were positive for *Hantavirus*.[1,3]

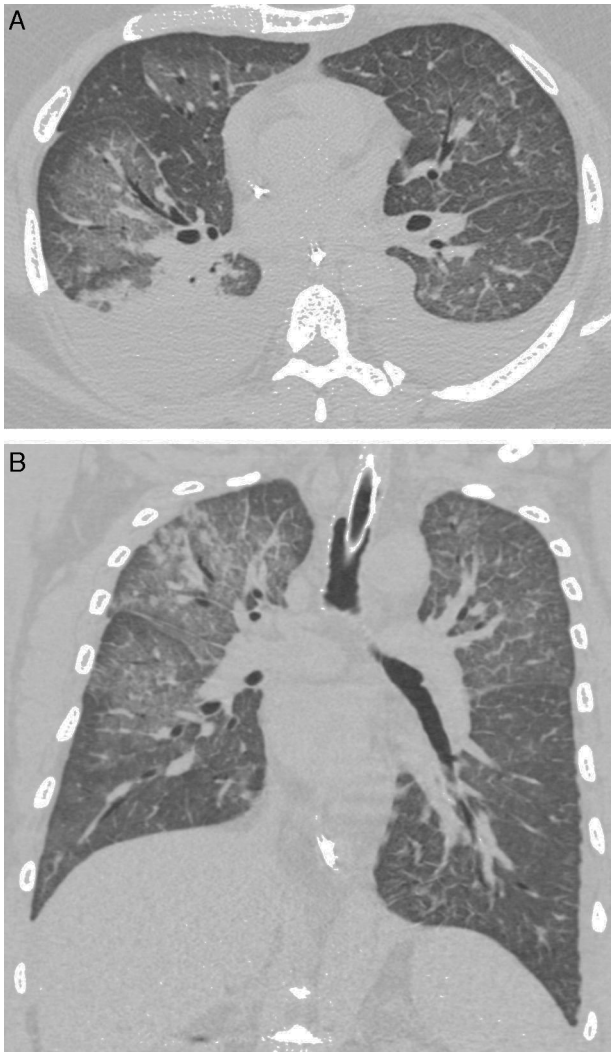

Fig. 1. A 28-year-old man with fever and rapidly progressive dyspnea. High-resolution computed tomography with axial (A) and coronal (B) reconstructions shows bilateral ground-glass opacities. Note also bilateral pleural effusion.

In conclusion, the predominant HRCT findings in patients with HPS were GGOs and smooth septal thickening. In the appropriate clinical setting, these findings are of great help in the diagnosis. Pleural effusion and peribronchovascular thickening are also frequent, but less characteristic findings.

References

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A Lung Lavage Technique in an Infant with Pulmonary Alveolar Proteinosis☆

Técnica de lavado pulmonar en lactante con proteinosis alveolar

To the Editor:

The treatment of choice for alveolar proteinosis in children is whole-lung lavage, by which proteinaceous material deposited in the alveoli is removed by instilling saline solution directly into the lung.[1] This procedure, which is rarely performed in the pediatric setting and for which no specific equipment has been developed, is a true technical challenge.[2]

We report the case of a female infant who was healthy until the age of 7 months, when she was diagnosed with acute myeloblastic leukemia type M5. She received a haploidentical stem cell transplant (SCT) from her father at the age of 13 months in her first full remission. She developed a post-SCT complication of generalized host-versus-graft disease, requiring intensive immunosuppressive treatment. Two months after the SCT, a chest computed tomography (CT) performed for persistent fever was normal.

She developed respiratory disease at the age of 20 months (7 months post-SCT), following *Klebsiella pneumoniae* sepsis, with progressive breathing difficulties and hypoxemia.

As her respiratory symptoms failed to improve despite antibiotic therapy, a chest CT was performed that showed a bilateral "crazy-paving" appearance. The patient was anesthetized and a simultaneous bronchoalveolar lavage (cytology showed abundant, dense granular material, PAS staining positive; *Pseudomonas aeruginosa* was grown on culture) and lung biopsy via mini-thoractomy were performed. This allowed us to rule out infection and interstitial lung disease due to surfactant deficit, and to confirm the diagnosis of alveolar proteinosis.

Given these findings, we decided to perform therapeutic lung lavage. A total of 2700 ml of warm saline solution was delivered to the right lung in 12 aliquots of 27 ml/kg. Two weeks later, the procedure was repeated in the left lung, using around 2500 ml.

Given the unavailability of double-lumen tubes for children under 8 years old, we decided to introduce 2 endotracheal tubes using direct laryngoscopy:

- One, 3.5 mm in diameter, was placed in the trachea to maintain ventilation.
- The other, 3 mm in diameter, with a balloon, was placed in one of the main bronchi, and telescoped until it reached the correct length and caliber; this one was used for the instillation of serum (Fig. 1A).

The correct placement of both tubes was confirmed with flexible bronchoscopy and fluoroscopy.

Fluid was introduced and removed from the bronchus by gravity; the tubes were alternately clamped to allow entry or exit of fluid (Fig. 1B).

After each procedure, the patient was admitted to the intensive care unit for 12 h. She received corticosteroids to prevent laryngeal edema and was extubated after a few hours without complications.

Clinical response after lavage of both lungs was very good, and her hypoxemia resolved rapidly. Two weeks after the procedure, a high-resolution, low-radiation CT of the chest was performed, revealing persistent ground-glass lung lesions, but to a lesser extent than before.

The presence of autoantibodies against granulocyte monocyte-colony stimulating factor (GM-CSF) in blood was ruled out, and GATA2 mutation testing was also normal.

Five months after the procedure, lung lavage was repeated due to reappearance of hypoxemia, and the patient again showed clinical improvement.

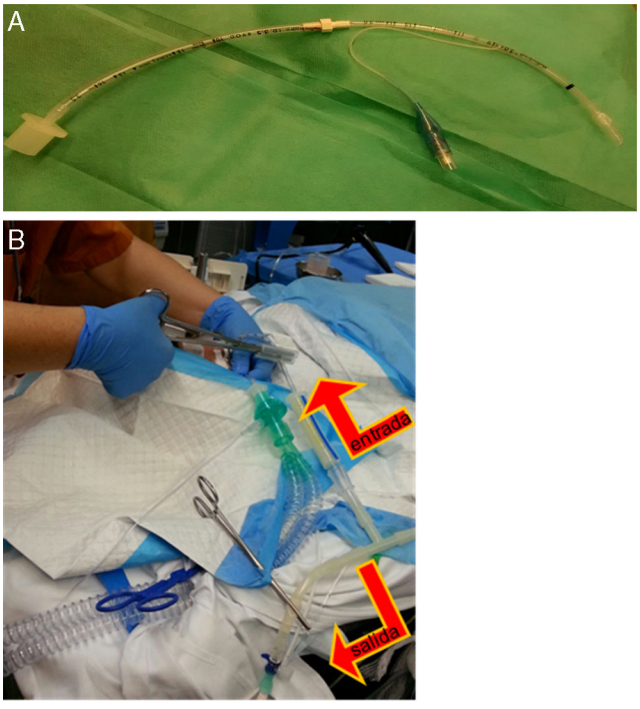

Fig. 1. (A) Endotracheal tube, 3 mm with balloon, extended by connecting it with another 3.5 mm tube, through which the warm saline solution was instilled and collected after it was placed in the main bronchus. (B) Image showing the tube located in the trachea, used to ventilate the patient, and the tube placed in the main bronchus, used to instill the saline solution with an alternating clamping system (the outflow was clamped at the time of instillation and the inflow was clamped at the time of emptying the lung fluid), indicated by the arrows.

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