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Acute respiratory distress syndrome secondary to human metapneumovirus infection in a young healthy adult

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Dear Editor,

There is growing interest in human metapneumovirus (hMPV) lung infection in intensive care unit (ICU). hMPV is a respiratory pathogen first discovered 10 years ago [1], but despite increasing data, the complete epidemiological features of hMPV remain largely unknown. In your journal, Vanspauwen et al. [2] reported six cases of hMPV detection in critically ill patients suspected of having hospital-acquired pneumonia. Recently, a case of severe hMPV lung infection in a pregnant woman was reported [3], and hMPV was identified in 18 % of patients hospitalized for severe pneumonia in ICU [4]. It is generally known that adults with underlying cardiopulmonary disease or immunocompromised patients are susceptible to hMPV infection [5]. However, we report a case of acute respiratory distress syndrome (ARDS) secondary to hMPV in a healthy woman.

A 59-year-old woman without relevant medical history was admitted to the ICU for acute dyspnea. Vital signs on admission were: heart rate 96/min,

blood pressure 105/73 mmHg, Glasgow Coma Scale 15, tympanic temperature 40 °C, respiratory rate 40/min, and oxygen saturation 83 % on room air. Physical examination revealed bilateral crackles and right bronchial breath sounds. Blood gases were pH 7.60, PaCO₂ 23 mmHg, PaO₂ 43 mmHg, and HCO₃⁻ 22 mmol/l. The patient rapidly developed severe respiratory failure unresponsive to oxygen therapy and required intubation and mechanical ventilation. Chest radiography showed right alveolar consolidation and bilateral interstitial infiltrates. The initial PaO₂/FiO₂ was 89 mmHg with lung ventilator protective settings [tidal volume (TV) 6 ml/kg, positive end-expiratory pressure (PEEP) 6 cmH₂O adjusted for plateau pressure ≤30 cmH₂O, FiO₂ 0.8]. Tests for pneumococcal and legionella urinary antigens were negative. Bacterial blood cultures (collected before the antibiotic treatment), bronchotracheal fluid cultures, serological tests for intracellular bacteria (*Legionella pneumophila*, *Coxiella burnetii*, *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*), and serological testing for extrinsic allergic alveolitis (bird breeder's and farmer's lung) were also negative. Therefore, an expanded panel of respiratory viruses was assessed on nasopharyngeal aspiration using immunofluorescence testing, and hMPV was detected (respiratory syncytial virus, parainfluenzae virus type 1, 2, 3, adenovirus, and influenza virus type A and B were negative). Diagnosis of hMPV lung infection was then confirmed by molecular testing. The overall duration of mechanical ventilation was 7 days. The patient was eventually discharged from hospital on day 23.

The originality of our observation is the severity of the respiratory failure and the absence of underlying disease. With the availability of new

diagnostic tools and the 2009 influenza A pandemic, attention is turning to the important role of respiratory viruses as a cause of severe pneumonia. Thus, hMPV diagnosis may have been missed as a causative agent in the past, when culture-independent microbiological techniques were not in use. We detected hMPV in the lower respiratory tract during respiratory failure, but we cannot confirm the fact that the presence of hMPV represented a genuine infection. However, accumulating data argue in favor of real hMPV pathogenicity [2, 3].

In conclusion, our observation provides further evidence that hMPV infection could have caused or contributed to ARDS in healthy adult. The role of hMPV in severe respiratory infection requires additional investigation.

Conflicts of interest No financial arrangements to disclose.

Ethical standard Written informed consent was obtained from the patient.

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