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Imaging of lung inflammation during severe influenza A: H1N1

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Sir: A 52-year-old man, a non-smoker and previously healthy, presented to the hospital with fever, cough and dyspnea. A nasal swab tested with real-time polymerase chain reaction showed a positive result for H1N1, and treatment with oseltamivir was started. Once transferred to the intensive care unit (ICU), he was diagnosed with acute respiratory distress syndrome (ARDS) and intubated. Bacterial cultures of tracheal aspirate and blood remained negative throughout his ICU stay; for this reason, we assume that a viral pneumonia from the influenza A-H1N1 virus was the cause of ARDS. Five days after intubation (six from symptoms onset), the patient was still markedly hypoxemic ($\text{PaO}_2/\text{FiO}_2$ ratio of 148 mmHg on a positive end-expiratory pressure of 18 cmH_2O) while ventilated with a protective ventilatory strategy (tidal volume 5.2 ml/kg PBW, plateau pressure 27 cmH_2O). In the context of a clinical trial ongoing in our center, he underwent a positron emission tomography (PET) scan with [^{18}F]fluoro-2-deoxy-D-glucose combined with computed tomography (CT). As shown by Fig. 1, the PET

scan disclosed an abnormally high uptake of [^{18}F]FDG, which, in the course of lung inflammatory processes, has been shown to indicate the presence of activated neutrophils [1, 2]. The increased [^{18}F]FDG uptake not only involved the areas characterized by a loss of aeration on the CT scan (black arrow, standardized uptake value 11.2), but also areas with preserved aeration, or showing only a modest ground-glass opacification (white arrows, SUV 6.8). The patient recovered; he was extubated

after 8 days and discharged alive from the ICU.

The presence of inflammation in normally aerated lung regions has already been shown in patients with ARDS [3] or in patients at risk for ARDS and subsequently developing the disease [4]. Thus, although the pattern observed in this patient does not seem specific to infection from influenza A, we believe that the image conveys the message well that the virus can trigger an extremely intense inflammatory

Fig. 1 Positron emission tomography (*upper panel*), computed tomography (*middle panel*) and fusion image (*lower panel*) of a patient affected by severe influenza A. The uptake of [^{18}F]FDG is abnormally high, not only in the areas characterized by a loss of aeration on the CT scan (*black arrow*), but also in the areas with preserved aeration (*white arrows*)

response, which, despite the use of a “protective ventilatory strategy,” quickly propagates throughout the lung.

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