

Acute eosinophilic pneumonia due to mercury vapor inhalation: a case report

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We present a case of a 52-year-old female who complained dyspnea and fever that developed 3 days ago. Chest x-ray and computed tomography (figure A,B) showed diffuse ground glass opacities in both upper lobes. She burned talismans a few days ago and inhaled smoke from the burned talismans. Bronchoalveolar lavage fluid showed elevated eosinophil count (24 %) and her blood test showed high levels of mercury (120.9 µg/L (Hg normal range ≤ 9.0 µg/L, Toxic: ≥ 50.0 µg/L). She was diagnosed with acute eosinophilic pneumonia caused by mercury vapor inhalation. When making talisman, mercury-containing inks are used. For cultural reasons, an accident occurs in which the mercury is vaporized and aspirated while the talisman is burned, and the vaporized mercury damages major organs including respiratory system. She treated with steroids and her dyspnea and lung infiltrations were improved without sequelae (figure C). It is the first report of acute eosinophilic pneumonia caused by mercury vapor inhalation.

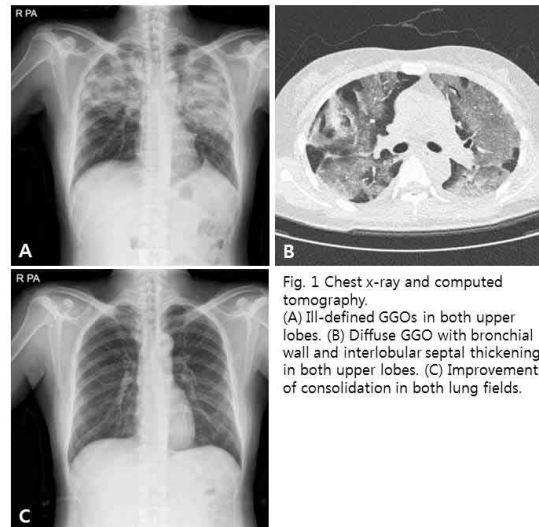


Fig. 1 Chest x-ray and computed tomography. (A) III-defined GGOs in both upper lobes. (B) Diffuse GGO with bronchial wall and interlobular septal thickening in both upper lobes. (C) Improvement of consolidation in both lung fields.

Risk factors for adverse events of muscle paralysis in patients under mechanical ventilation

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Background/Aims: Some patients who undergo muscle paralysis during mechanical ventilation (MV) treatment have a progress toward development of severe respiratory acidosis. They sometimes encounter hemodynamically instability. Contrary to a well-known knowledge of long-term adverse outcomes of neuromuscular blocking agents (NMBA), respiratory acidosis immediately after NMBA infusion has not been reported. We investigated to reveal the risk factors for post-NMBA respiratory acidosis in patients under mechanical ventilation who started cisatracurium just before the event. **Methods:** Following a retrospective review of the medical records of patients admitted to the ICU from July to October 2017, we included adults who received cisatracurium during MV. Data of vital signs, arterial blood gas analysis (ABGA), and parameters of the ventilator before and after NMBA infusion were recruited. Respiratory acidosis developed after cisatracurium infusion was defined discretionally as decreased arterial pH more than 0.2. **Results:** The mean age of the patients was 62 years and 64% (n= 57) was male. Of total 89 Patients, nine patients (10%) showed a progression of acidosis. Among them, three patients' blood pressure dropped immediately after NMBA infusion and vasopressor was needed. In univariate analysis, a higher pH, lower arterial partial pressure of carbon dioxide (PaCO₂), and higher pulse rate (PR) measured before NMBA infusion were associated with progression of respiratory acidosis. On the other hand, underlying lung disease, disease severity, and pre-NMBA tidal volume based on patients' efforts are not related with a progression of respiratory acidosis. In multiple logistic regression model, lower PaCO₂ (OR, 0.87; 95% CI 0.76–0.99) and higher PR (OR, 1.05; 95% CI 1.01–1.09) are associated with progression of respiratory acidosis after NMBA infusion. **Conclusions:** Lower PaCO₂ and higher PR are risk factors for post-NMBA respiratory acidosis. It suggested patients with respiratory failure who showed active cardiopulmonary compensation might have a risk for a progression of respiratory acidosis after muscle paralysis.

Table 1. Risk factors for progression of respiratory acidosis after infusion of neuromuscular blocking agent

Variables	OR	95% CI	p-value
Age	0.972	0.908 – 1.041	0.419
Sex			0.852
Male	Reference		
Female	1.256	0.115 – 1.141	
Pre-NMBA PaCO ₂	0.870	0.757 – 0.999	0.049
Pre-NMBA pulse rate	1.049	1.006 – 1.094	0.024

OR, odd ratio; CI, confidence interval; NMBA, neuromuscular blocking agent; PaCO₂, arterial partial pressure of carbon dioxide