Acute Eosinophilic Pneumonia Induced by Varnish Particles: A Diagnostic Challenge

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Acute eosinophilic pneumonia (AEP) is a rare disease of unknown cause. Unlike chronic idiopathic eosinophilic pneumonia, the disease mostly affects males with no history of asthma or allergies [1]. Several types of exposure, such as a recent change in tobacco consumption, are thought to be responsible for AEP [2]. Given that the clinical presentation of AEP is nonspecific (cough, fever, pleural effusion), the condition can often be mistaken for acute infectious pneumonia or acute respiratory distress syndrome (ARDS) [3]. The key investigation is bronchoalveolar lavage (BAL), which confirms AEP by revealing an eosinophilic cell pattern (>25% eosinophils) in the differential cell count of BAL fluid. We report a case of AEP induced by domestic exposure to varnish particles and illustrate the difficulty in diagnosing this condition correctly.

A 57-year-old man with no medical history of interest presented to the emergency department with a 2-day history of chest pain, dry cough, and progressive dyspnea. He had no history of smoking, substance use, or allergy. He was in excellent physical condition and exercised every day. Ten days before his first respiratory symptoms, he had been exposed for several days to varnish particles without respiratory protection in a confined environment (wooden door maintenance). Twenty-four hours before admission, his family doctor had prescribed prednisone for flu-like syndrome.

On examination in the emergency department, he was febrile with dyspnea at rest and oxygen saturation of 87% in ambient air. Auscultation revealed bilateral bronchial sounds with crackles. Thoracic computed tomography (CT) revealed interstitial syndrome (interlobular septal thickening) and bilateral ground-glass pattern with bilateral basal condensations. Biological tests revealed inflammatory syndrome. Kidney and liver function were normal. Intravenous cefotaxime and spiramycin were initiated for suspected atypical pneumonia. On the seventh day after admission, the patient was intubated for mechanical ventilation owing to hypoxemia. A second thoracic CT scan carried out on day 8 revealed worsening of the previous abnormalities

and a mild right pleural effusion (Figure). BAL revealed 2.7×10^5 cells/mL with 75% neutrophils, 10% macrophages, and 0% eosinophils. Blood tests ruled out an autoimmune cause, and microbiological samples were negative. Antibiotic therapy was switched to piperacillin-tazobactam, and the severity of the patient's condition ($PaO_2/FiO_2 = 90$) led to 2 periods of prone positioning.

Because of the persistence of hypoxemia, a second BAL was performed on day 14 and revealed eosinophilic alveolitis (6.9×10^5 cells/mL with 40% eosinophils). We started intravenous corticosteroids on day 14 (1.5 mg/kg/d of methylprednisolone). The patient's health improved dramatically, enabling weaning from mechanical ventilation within 4 days and oxygen therapy before admission to the pulmonology department. Parenchymal opacities had completely disappeared after 5 days of corticosteroids. There was no relapse during the follow-up period, and pulmonary function test results were normal at discharge.

Several environmental triggers have been described in AEP (exposure to smoke from fireworks [4] and dust after the attacks on the World Trade Center [5]). However, as far as we know, this is the first report of AEP induced by domestic exposure to varnish particles. The diagnosis of AEP was confirmed by eosinophilic alveolitis (40% eosinophils) in the second BAL and by the full recovery achieved with corticosteroids, as expected in AEP [6]. Peripheral eosinophilia was never observed, as is often the case in AEP, which differs significantly from chronic eosinophilic pneumonia [7]. This finding is important when ruling out drug-induced AEP, because all reported cases involve considerable peripheral eosinophilia [8]. Nevertheless, it is important to note that the initial outpatient prescription of oral corticosteroids may explain the normal white blood cell count. Exposure to varnish was confirmed by the patient himself, who reported 7 days of intense exposure in a confined environment without respiratory protection. The time lapse of 10 days between the first exposure and the first respiratory symptoms is consistent with this etiology. The main toxic chemical component in the varnish was isocyanate, which has been recognized for more than 60 years as a common cause of sensitization leading to various pulmonary diseases such as occupational asthma. It should therefore be taken into account when AEP is suspected. The other chemical compounds in this case (alkyls) are unlikely to be involved in triggering eosinophilic lung diseases. The patient declined to undergo allergy tests. The exact pathophysiology of AEP is unclear, although hypersensitivity has been reported to be a possible mechanism, and the acute onset and striking response to corticosteroids clearly favor this mechanism [9]. The absence of an eosinophilic cell pattern in the first BAL fluid sample is unusual in AEP. Initially, BAL fluid showed a neutrophilic cell pattern, as reported in cigarette-induced AEP [10]. It is therefore important to repeat BAL, in case severe hypoxemia is unresponsive to the conventional treatment of community-acquired pneumonia or ARDS, in order not to miss the differential diagnosis and, in particular, AEP.

Finally, we considered other etiologies of eosinophilic pneumonia in this case. Drug-induced AEP is unlikely in the light of the antibiotics timeline, even though some cases have been induced by other antibiotics [8]. Prompt recovery after corticosteroids were started, with maintenance of antibiotics, rules out this diagnosis. Moreover, there was no argument in favor of a parasitic disease, hematologic disease, or hypereosinophilic syndrome.

To conclude, we report a case of AEP induced by domestic exposure to varnish particles containing isocyanate in a 57-year-old man with no medical history of interest. Diagnosis was made only after a second BAL. We emphasize the need to repeat BAL for differential cell count analysis in cases of severe hypoxemia that are refractory to conventional treatment. AEP is similar to community-acquired pneumonia and ARDS in terms of clinical and radiological presentation, and the eosinophilic cell pattern may be absent in some cases or at the initial stage of AEP, especially if the patient has received corticosteroids.

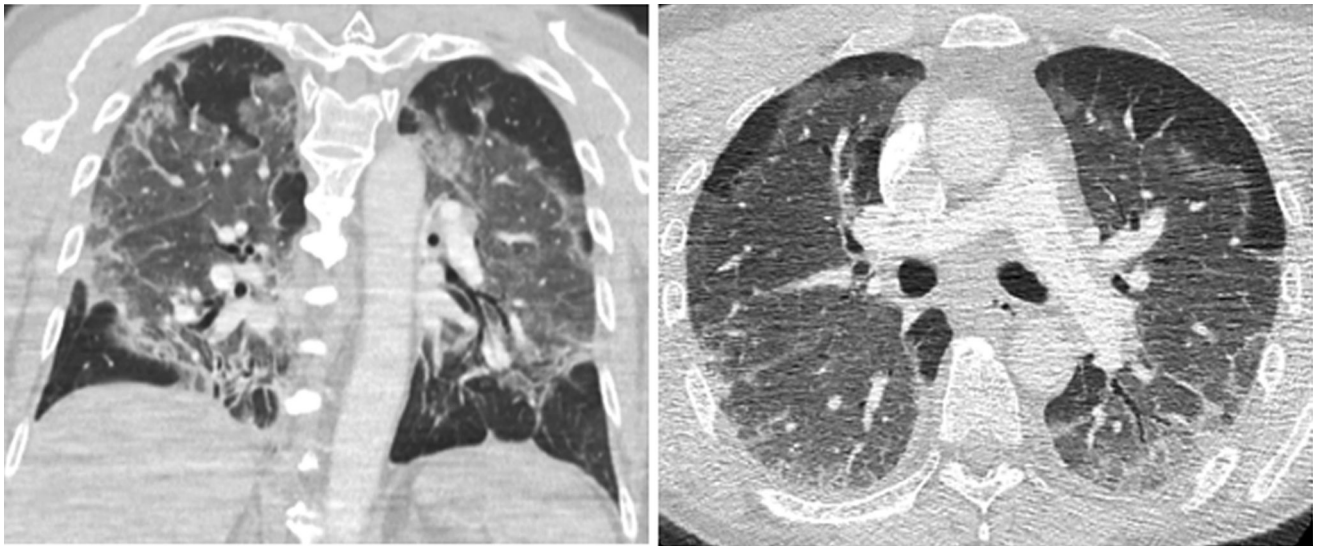

Figure. Thoracic computed tomography scan on day 8 after admission to the intensive care unit revealing worsening of ground-glass opacities.

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Conflicts of Interest

The authors declare that they have no conflicts of interest.

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