A case of eosinophilic pneumonia simultaneously diagnosed in a patient and a tame cat: a case report

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Abstract

Introduction: Chronic eosinophilic pneumonia is an idiopathic disorder of unknown etiology. Corticosteroid treatment provides a good response but recurrence frequently occurs after tapering of corticosteroid. Chronic eosinophilic pneumonia occurs predominantly in middle-aged women and non-cigarette smokers, which leads to the speculation that environmental antigens, particularly in the home, contribute to the etiology.

Case presentation: A 66-year-old Japanese woman was given a diagnosis of chronic eosinophilic pneumonia for 8 years and was treated with prednisone. She developed respiratory symptoms again with tapering of prednisone (10mg/day). A chest radiograph revealed patchy shadows in her bilateral upper lung fields, and bronchoalveolar lavage fluid revealed marked eosinophilia. Based on negative findings for other causes of eosinophilia, the diagnosis of the recurrence of chronic eosinophilic pneumonia was established. She was treated with prednisone (20mg/day), which demonstrated rapid improvement. Around the same time, her tame cat developed oral breathing, tachypnea and peripheral eosinophilia. Chest radiography of the cat revealed ground-glass opacity in its bilateral upper lung fields. Eosinophilic pneumonia was also diagnosed in the cat that was treated by prednisone (3mg/day). Since eosinophilic pneumonia was diagnosed simultaneously in the patient and her tame cat, it can be suggested that inhaled environmental antigens in the home caused the eosinophilic pneumonia. After moving out of her home, she and the cat had no recurrence of eosinophilic pneumonia.

Conclusions: Although chronic eosinophilic pneumonia is an idiopathic disorder of unknown etiology, our case suggests that inhaled environmental antigens in the home may be associated with the causes of chronic eosinophilic pneumonia. A pet’s disease may give us an important clue for the therapeutic approach of the owner’s disease.

Keywords: Cat, Eosinophilic pneumonia, Inhaled environmental antigens
respiratory alkalosis (pH 7.55; partial pressure of oxygen in arterial blood, 87.7 mmHg; partial pressure of carbon dioxide in arterial blood, 30.3 mmHg; and bicarbonate, 30.3 mEq/L). Her leukocyte count was 7200/ul; 4% were eosinophils. Results of routine serum chemical studies were almost normal except for serum C-reactive protein of 1.1 mg/mL (normal, <0.3 mg/mL). Antineutrophil cytoplasmic antibodies were absent. Her serum immunoglobulin E (IgE) level was 56.4 IU/mL (normal, <173 IU/mL). Specific IgE to house dust including Dermatophagoides pteronyssinus or Dermatophagoides farinae, house dust mites in Japan, and Aspergillus fumigatus were all negative (radioallergosorbent tests; class range 0). Her urine analysis was normal, with no active sediments. Stool examinations for ova or parasites were negative. A pulmonary function test demonstrated an obstructive pattern with reduced diffusing capacity of her lungs for carbon monoxide (DLCO) (forced vital capacity, 106%; forced expiratory volume, 64%; DLCO, 66%). A chest radiograph revealed patchy shadows in her bilateral upper lung fields (Figure 1A). A chest computed tomography showed bilateral patchy ground-glass opacities with subpleural predominance (Figure 1B). Bronchoalveolar lavage fluid recovered 980,000 cells per mL with a differential 62% of eosinophils. Based on negative findings for other causes of eosinophilia, the diagnosis of the recurrence of CEP was established. She was treated with prednisone, 20 mg/day, which demonstrated rapid improvement of her clinical picture and chest imaging (Figure 2).

Around that same time, her tame cat of 10 years, developed oral breathing, tachypnea (40 breaths/minute) and peripheral eosinophilia (12%). Chest radiograph of the tame cat revealed ground-glass opacity in its bilateral upper lung fields (Figure 3). The tame cat, which had lived in the same house as the patient for 8 years, was also diagnosed as having eosinophilic pneumonia and was treated with 3 mg/day (1 mg/kg) of prednisone for 3 days, resulting in a rapid improvement of the clinical picture. Since eosinophilic pneumonia was diagnosed simultaneously in the patient and the tame cat, it can be suggested that inhaled environmental antigens in the home caused the eosinophilic pneumonia. After moving out of her home, she had no recurrence of CEP with tapering of prednisone. The tame cat also had no recurrence of eosinophilic pneumonia without prednisone.

**Discussion**

Because fewer than 10% of patients with CEP improve spontaneously, corticosteroid therapy should be indicated. Most patients undergo prolonged corticosteroid therapy due to common recurrence after cessation or tapering of corticosteroid therapy.

Concerning the etiology, the presence of asthma or the history of atopy accompanies or precedes CEP in over 50% of cases, suggesting that CEP is a chronic hypersensitive reaction related to allergic disorders [1].
CEP occurs predominantly in middle-aged women and non-cigarette smokers, which leads to the speculation that environmental antigens, particularly in the home, contribute to the etiology of CEP. Environmental inhaled antigens often induce acute eosinophilic pneumonia without recurrence of disease because it is easy to detect and avoid antigens such as cigarette smoke [2,3], dust [4], or spiders [5]. However, in cases of eosinophilic pneumonia induced by environmental antigens in the home, recurrence is likely to occur with the tapering of corticosteroid if patients do not move out of their home. In the present case, the inhalation of environmental antigens present in the home might have caused the CEP, based on the negative findings of other causes of pulmonary eosinophilic syndrome and the absence of recurrence after she moved to a different home. Environmental antigens in the home can be considered in the etiology of CEP for not only the patient but also the cat, although it is speculative due to the unidentified antigens and no provocative challenge.

Conclusions
Although CEP is an idiopathic disorder of unknown etiology, our case suggests that inhaled environmental antigens in the home may be associated with the causes of CEP, and that a pet’s disease may give us an important clue for the therapeutic approach of the owner’s disease.

Consent
The authors obtained written informed consent from the patient to publish this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Abbreviations
CEP: Chronic eosinophilic pneumonia; DLCO: diffusing capacity of the lung for carbon monoxide; IgE: immunoglobulin E.

Competing interests
The authors declare that there are no competing interests regarding the publication of this case report.

Authors’ contributions
TT and MK drafted the manuscript and performed the literature search. JT provided guidance for drafting the manuscript. RK and ET participated in its design and coordination. All authors read and approved the final manuscript.

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