Successful Management of Eosinophilic Bronchopneumopathy in a Dog

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Abstract A dog was presented with a cough, dyspnea, nasal discharge, gagging, and exercise intolerance. The dog showed leukocytosis, peripheral eosinophilia, and an increase C-reactive protein. The radiographic findings noted bronchointerstitial infiltration, intrathoracic lymphadenopathy, and soft tissue opacity mass. Computed tomography findings showed thickening of the bronchus and bronchiole. Also, peri-bronchial consolidation and generalized intrathoracic lymphadenopathy was present. On blind bronchoalveolar lavage and pulmonary cytology, there were significantly increased eosinophils. Canine pulmonary respiratory pathogens from a real-time polymerase chain reaction analysis was negative. Consequently, the dog was diagnosed with eosinophilic bronchopneumopathy. Clinical signs improved significantly within a few days after treatment with an oral corticosteroid.

Key words eosinophilic bronchopneumopathy, bronchoalveolar lavage, corticosteroid, dog.

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Introduction

Eosinophilic bronchopneumopathy (EBP) is a broad term characterized by inflammatory lung and bronchial mucosa disease in which the predominant infiltrating cell is the eosinophil. The disease is considered a hypersensitivity response (5). The mechanisms and underlying antigen source of pulmonary hypersensitivity are poorly understood in human and veterinary medicine (3). Considerations in humans and animals include bacteria, fungi, pulmonary parasites, heartworms, drugs, neoplasia, and inhaled allergens. However, in many cases, the inciting antigens are not found (2). This case report describes the clinicopathologic, cytology, imaging features, and successful management in one dog with EBP.

Case Report

A 7-year-old, spayed female mixed dog, weighing 2.45 kg, was referred with a 2-week-history of coughing, dyspnea, tachypnea, exercise intolerance, nasal discharge, and gagging. There had been no response to 14-day prescription of pimobendan (0.25 mg/kg, PO, q24h), furosemide (0.5 mg/kg, PO, q24h), enalapril (0.2 mg/kg, PO, q24h), spironolactone (0.25 mg/kg, PO, q24h), and theophylline (5 mg/kg, PO, q24h). This is because the local clinic misunderstood the bulging of left atrium on the right lateral thoracic radiograph and judged it to be a symptom of heart disease. At the initial presentation, the patient was slightly depressed and had an obvious tachypnea characterized by rapid and shallow breathing. The lung sound was normal, and the dog had no cardiac murmur. Mild leukocytosis (15,360 cells/mcL, RR: 5,200-13,900), peripheral eosinophilia (5,210 cells/mcL, RR: 0-600) and increase C-reactive protein (4.83 mg/L, RR: 0-2) was detected. The heartworm IDEXX 4Dx kit was negative.

On thoracic radiography, a soft tissue opacity nodule was found in the left caudal lung lobe. Also, a diffuse bronchial pattern was found on lateral radiography. An enlargement of the tracheobronchial, sternal, cranial mediastinal lymph node was suspected (Fig. 1). The differential diagnosis considered for these conditions included primary solitary lung tumor, benign lesion (lung abscess or granuloma), lung haematocoeles or cyst, and lobar pneumonia.

An additional computed tomography (CT) examination was performed to narrow down the differential diagnosis. The CT findings showed an amorphous soft tissue structure that was observed in front of the bronchial area of the trachea, branching to the left caudal lung lobe; it was observed with homogeneous contrast enhancement. The bilateral lung lobes appeared to be contrast enhancement with ground-glass opacity, and an air bronchogram was observed between the parenchyma. The thickness from left and right of the bronchial branching into the caudal lung lobes to the terminal bronchial was observed to be very thick compared to normal, and mucus clogged the bronchial lumen in the right caudal lung lobe. Intrathoracic lymphadenopathy was also present. Sternal, cranial mediastinal, left, and middle tracheobronchial lymph nodes were considered enlarged (Fig. 2). All lymph nodes except the middle tracheobronchial lymph node were observed with homogenous contrast enhancement. The left and right bronchi, which branch to the rear of the trachea and carina, were observed to be displaced or pressed to the right and ventral due to the enlargement of tracheobronchial lymph nodes. Also, the azygous vein and aorta were displaced to the dorsal side.

Cytology from the bronchoalveolar lavage fluid (BALF) and fine-needle aspiration (FNA) from left caudal lung lobe soft tissue opacity was performed. The percentage of inflammatory cells from BALF was eosinophil 68%, neutrophil 1

Fig. 1. Dorsoventral (A) and right lateral (B) radiographs of thorax in the case. Soft tissue opacity nodule was found in the left caudal lung lobe (A). A diffuse bronchial pattern was found on lateral radiography (B). Tracheobronchial, sternal, cranial mediastinal lymph node enlargement was suspected (B).

Fig. 2. CT image of cranial lung lobe. Amorphous soft tissue structure is observed in front of the bronchial area of the trachea branching to the left caudal lung lobe (A). Wall thickness from left to right bronchi and plugging of the bronchial lumen by mucus in the right caudal lung lobe was observed (B). Intrathoracic lymphadenopathy was found (C).

7%, lymphocyte 8%, and monocyte 7% (Fig. 3). Also, the percentage of inflammatory cells from the soft tissue opacity region was eosinophil 68%, neutrophil 26%, monocyte 4%, and lymphocyte 2%. BALF was tested to rule out respiratory infections. The test result from the canine respiratory pathogens real-time polymerase chain reaction (PCR) analysis from POBANILAB laboratory were all negative; there are canine distemper, parainfluenza virus 3, respiratory corona virus, influenza virus, adenovirus type II, herpes virus, influenza A/B, streptococcus zooepidermicus, aspergillus, mycoplasma cynos, bordetella bronchiseptica, cryptococcus spp, pneumocystis carinii, histoplasma, and blastomyces disease in the PCR analysis.

EBP was diagnosed by based on these findings. There had been no response to fenbendazole (50 mg/kg PO q24h for 3 days). Treatment with prednisolone (1 mg/kg PO q12h) was conducted for 1 week. On follow-up, the owner reported that the patient's cough, dyspnea, exercise intolerance were almost gone, but there was still a little the nasal discharge. And, the peripheral blood eosinophil counts returned to the normal range. On thoracic radiography, the abnormalities on pulmonary parenchyma were improved significantly and there was no soft tissue opacity on the left caudal lung lobe (Fig. 4). However, the patients appeared to have a little bit of weakness, polyuria, polydipsia, and polyphagia. After maintaining the current dose for another week, the symptoms improved significantly, and the treatment slowly reduced the dosage to prednisolone 0.5 mg/kg every other day for two to three weeks.

Fig. 3. Blind bronchoalveolar lavage cytology of this case. The percentage of eosinophils was more than 60% in BALF cytology.

Discussion

The cause and mechanisms of EBP are poorly understood in human and veterinary medicine. However, hypersensitivity may be associated with the disease (1,2). And, dogs affected with EBP are usually young adults (4-6 years) and the disease is found in any breed of dog and any size.

Diagnosis usually depends on radiographic and bronchoscopic findings, peripheral eosinophilia, eosinophilic infiltration demonstrated by cytology of BALF, or histology of tissue biopsy (1,2). The most common radiographic findings of EBP are bronchial and bronchointerstitial patterns topographically distributed to at least the caudodorsal lung field. Furthermore, a bronchointerstitial pattern predominates within the caudodorsal lung field (4).

CT findings are a valuable tool to evaluate suspected EBP

Fig. 4. Dorsoventral (A) and right lateral (B) radiographs of thorax in the case after 1 week of treatment (prednisolone 1 mg/kg, PO, q12h). Reduction of lymphadenopathy and bronchial pattern was observed.

and to rule out other underlying diseases like pulmonary neoplasia.

In this case, the patient was misdiagnosed with cardiac disease due to left atrium bulging sign on the lateral radiograph in a local clinic. The response to pimobendan, furosemide, enalapril, spironolactone was poor. The patient had no cardiac murmur and peripheral eosinophilia, so we thought the differential diagnosis was pulmonary granuloma, pneumonia, infections, or pulmonary neoplasia. But, the absence of peripheral blood eosinophilia did not exclude a diagnosis of EBP (2). Because it was a small dog, the bronchoscopy did not fit the ET tube size, the lumen could not be identified through the bronchoscope, but the airway and pulmonary eosinophilia could be identified through a blind BAL. EBP is characterized by an increased percentage of eosinophils and neutrophils in BALF, less than 5% eosinophils are usually found in the BALF from healthy dogs (2). In this case, eosinophils and neutrophils percentage of BALF (68%, 7%, respectively) were significantly increased. Based on the CT findings, the cytology from BALF and tissue FNA and PCR analysis, differential diagnosis narrowed eosinophilic EBP or eosinophilic pulmonary granuloma (EPG). Although both diseases may show eosinophilic airway inflammation and peripheral eosinophilia, EPG differs from EBP in that the presence of pulmonary nodules and masses is composed of eosinophils, macrophages, and various combinations of inflammatory cells. Also, EBP has a clinically better response to immunosuppressive treatment (7). Some authors have suggested that some cases of EPG might be one of the advanced stages of EBP (6). EPG is strongly associated with a heartworm infection. In this case, the heartworm IDXX 4Dx kit was negative and the owner consistently administers heartworm medication every month. In addition, the clinical and radiography response to steroid therapy was perfectly good. Based on results, the case was diagnosed as an EBP.

Prednisolone was prescribed as treatment for 2 weeks. After 1 week, the cough, dyspnea, and exercise intolerance improved significantly, but the nasal discharge remained problematic. Nasal discharge is sometimes more resistant to treatment (1). As this may be part of hypersensitivity reaction, additional antihistamine medications with clemastine improved clinical signs. Prednisolone tapered to every other day for 2-3 weeks and discontinued. The patients have had a good quality of life and clinical signs do not recur from now.

The time from onset of clinical signs until the diagnosis does not affect response to therapy (1). Also, age at the time of diagnosis does not affect the response to therapy (2). The poorest responses to therapy occur in cases that are irregularly treated, with repeated abrupt cessation of high doses of medication without tapering (2).

In conclusion, the dog was diagnosed with eosinophilic bronchopneumopathy. Clinical signs improved significantly within a few days after treatment with an oral corticosteroid. The most important aspects of EBP management are correct diagnosis and client education. Therefore, veterinarians should educate the owner on their responsibility and committed care. Also, regular rechecking and gradual tapering of oral steroids are required.

Conflicts of Interest

The authors have no conflicting interests.

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