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A Case Report: Pulmonary Infiltrates with Eosinophilia in a Canine

Lisa DeNault
Iowa State University

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LISA DE NAULT†

Eosinophilic lung disease can be categorized as either pulmonary infiltrates with eosinophilia or eosinophilic pulmonary granulomatosis, two broad terms classifying inflammatory lung disease where eosinophils predominate.[1,4,5] Differential diagnoses for eosinophilic lung disease are chronic allergic bronchitis, primary parasitic lung disease, bronchopneumonia (bacterial or mycotic), diffuse infiltrative neoplasm, mononuclear granulomatous disease like systemic lupus erythematosis, infectious diseases like rickettsia, atypical bacterial, protozoa, fungal, parasitic, or secondary bacterial infection due to foreign body.[2,4,5]

Respiratory tract parasites such as Aelurostronglus, Paragonumus, Filaroides, and Capillaria species can cause peripheral and pulmonary eosinophilia.[5] Pulmonary infiltrates with eosinophilia (PIE) is usually thought of as an interstitial lung disease. Another common disease in cats is allergic bronchitis, which is also an eosinophilic pulmonary disease but primarily affects the airways rather than the interstitial parenchyma. PIE is a term that is descriptive rather than indicating a particular etiology.[1,4,6]

PIE is a rare disease in dogs that is probably due to an extreme hypersensitivity reaction of the lungs. Differential diagnoses for pulmonary nodules seen on radiographs would be fungal diseases or metastatic neoplasm. One needs to determine an inciting antigen which could be heartworm, pulmonary parasites, drug reactions, or inhaled allergens.[2,7] In most cases, the cause is unknown.[1] Although PIE is strongly associated with heartworm disease, many more dogs with PIE are heartworm-negative or live in areas where heartworm is not endemic like Chancey in California.[2]

Even though there are many potential causes for PIE, most cases are idiopathic.[2] There is no age, sex, or breed predilection for this disease.[1] Even though no predisposition has been shown, many more medium to large breed outside dogs are affected.[2] The clinical presentation usually involves slowly progressive respiratory signs with a chronic cough, difficulty breathing, and exercise intolerance.[4,6] Another clue is that the cough is unresponsive to antibiotics.[2,5] There may be signs of anorexia or decreased appetite and associated weight loss but many times these are only mild and unnoticed by the owners.[1-3] Diffuse bilateral crackles can be auscultated in the lungs on physical exam. There may be moist or dry rales, but often no rales at all are heard.[3,6]

Diagnosis of PIE is based on peripheral eosinophilia, interstitial lung pattern seen on thoracic radiographs with nodules with fuzzy borders, and elimination of any other causes for eosinophilia.[1,2,5,6] Radiographs are very helpful because one can see patchy alveolar opacities and lung consolidation with or without hilar lymphadenopathy.[1,2,5] The final diagnostic test is a tracheal wash showing predominantly eosinophilic inflammation.[1,2] Cytology is usually diagnostic. Other more aggressive diagnostics are bronchial alveolar lavage, lung aspiration, or lung biopsy.[1,3,4,5] Heartworm tests and fecal flotations should also be run on every cases to rule out heartworm disease and lung worms.

Case Study

Chancey, a 3.5 year-old spayed female mixed-breed dog, presented with acute respiratory distress to Animal Urgent Care on July 5, 1999. The patient had been quiet for a few days. Then, the day before presentation, the clients noticed her breathing became slightly heavy. She refused food. There was no known exposure to rodenticide, and previous medical problems. She did have a recent history of being groomed. They had recently moved but had not been out-of-state.

Upon physical exam, she exhibited marked respiratory distress. The patient's mucous membranes were red and her capillary refill time was 1-2 seconds. The RDVM

†Lisa De Nault graduates from the Iowa State University College of Veterinary Medicine in May 2000.

auscultated increased bronchovesicular sounds over all lung fields. Intercostal scalloping was seen but no cupula movement detected. The heart sounds were within normal limits. The RDVM noticed multiple pinpoint macules on the skin and the hair coat was thin. Her temperature was 96°F, with a pulse of 60 bpm, and a respiratory rate of 70 per minute.

Aggressive medical care was given immediately. Chancey was put on an oxygen flow via a mask. An intravenous catheter was placed in her left cephalic vein and morphine (0.25 mg/lb IV) was administered. A nasal canula was placed with the oxygen flow set at three liters per minute. Lasix was given IV at 1mg/lb.

Radiographs of the thorax showed a diffiuse alveolar interstitial lung pattern with concentration in the perihilar-caudal lobes. 800 mg cefazolin IV was given. Arterial blood gas showed PO_2 46%, PO_2 88mmHg, pH 7.1, and HCO_3 29. A second nasal canula was placed and oxygen mask continued. The repeated blood gases showed only minimal improvement in PO_2 at 69 and bicarbonate at 115. An emergency tracheotomy was performed under 7mg valium IV and isoflurane with mask induction. The pulse oximetry reading only increased to 86% post-tracheotomy.

All Care got the call around 10 am for an emergency pick up of Chancey; at this point I got the case. Chancey was picked up via emergency transport. Using IPPV for the trip back to All Care, I hand bagged with three liters per minute of oxygen. We tried to put her on a ventilator but the patient resisted because she was still awake, though in severe respiratory distress.

At noon, her oxygen saturation was only up to 80% on 100% oxygen and supplement IPPV. The primary rule outs at this point were heartworm disease, fungal infection, lung worms, and PIE. During the afternoon, I hand bagged her for four hours and the oxygen saturation never went above 80%. Day 1 continued with hand bagging for 10 hours.

She was put on Baytril and Ampicillin. Heparin therapy was started because of thrombocytopenia, and put in CCU on nasal oxygen and suctioning of tracheal tube every two hours.

On day 2, prednisone was added at 10mg per os twice a day. Brethine and nebulization were also added to the CCU orders.

On day 3, the Baytril and Ampicillin were continued, and the prednisone dose was increased to 20 mg per os twice a day. The brethine, nebuliation, and suctioning of the endotracheal tube were continued. Cytotec and zantac were added because of bloody diarrhea and vomiting reported overnight.

On day 4, Chancey had not improved greatly and respiratory difficulty continued. On day 5, the antibiotics were discontinued and nasal oxygen was discontinued because Chancey had greatly improved overnight. The tracheal tube was pulled late in the afternoon subsequent to vast radiographic improvement in the lung fields, which were almost clear of nodules and infiltrates.

Chancey was discharged on day 6 and sent home with Orbax and prednisone at 20 mg per os twice a day.

Radiographs taken at All Care showed a bronchialinterstital lung pattern. The primary differential diagnoses are: bronchial asthma, allergic bronchitis, thromboembolic pneumonitis, fungal infection, hemorrhage more alveolar, smoke inhalation, heartworm disease. Three days later radiographs were taken again and showed a bronchial alveolar and interstitial pattern. There was microcardia secondary to hypovolemia due to treatment with lasix.

Blood profiles were normal white blood cell count except for a peripheral eosinophilia. A tracheal wash was done and cytology came back as eosinophilic inflammation. Bacterial and fungal cultures were negative growth. Heartworm antigen test results were negative as expected since Chancey was a California dog with no history of traveling into known heartworm areas of the US.

Bacterial cultures showed no aerobic growth from direct plating and broth culture after 72 hours and no anaerobic growth after 6 days. Fluid cytology from the tracheal wash had adequate cellularity and primarily contain inflammatory cells with occasional cuboidal to columnar ciliated respiratory epithelial cells. The inflammatory cells primarily consisted of eosinophils with lesser numbers of neutrophils and occasional alveolar macrophages.

No infectious agents or evidence of neoplasia were observed. Cytologic diagnosis identified moderate eosinophilic inflammation consistent with a hypersensitivity reaction or

parasite infection. Fecal flotation and direct smear showed no ova or parasites including giardia. Blood work taken on July 5 reported back elevated eosinophils absolute count at 3724 or 28% (*normal range 0-1200*) and elevated basophils absolute count at 399 or 3% (*normal range 0-150*).

T3, T4, and free T4 were all report low detected by radioimmunoassay. Antinuclear antibody titer was less than 1:16, which is normal. Platelet count was low at 119,000 (normal range 170,000 to 400,000). Coombs cold and warm direct tests were both negative. Rheumatoid factor level was normal, prothrombin, activated prothrombin time, and fibrinogen quantitative levels were all normal.

Cryptococcal antigen was negative. Fungal serology and a cocci screen were negative for coccidioidomycosis, and fungal serology was negative for histoplasma, blastomyces, and aspergillus.

Final diagnosis was PIE with a potential for recurring episodes of respiratory distress. Upon discharge, the owners were instructed to continue giving Orbax 68mg tablets for seven days once daily, and 20 mg tablets of prednisone to give half a tablet twice daily for 14 days. The prednisone dose was then lowered to a half-tablet once per day for 14 days, and eventually down to a half-tablet every other day for 14 days.

Also suggested to the owners was to keep Chancey in a quiet, stress free environment and to monitor for respiratory difficulty, lethargy, loss of appetite, vomiting, or any other abnormal signs and to have a recheck in seven days.

To follow up at the seven day recheck, Chancey was bright, alert, and responsive. She was panting but not coughing, sneezing, vomiting, and didn't have any diarrhea. The patient's mucous membranes were pink and her CRT was less than 2 seconds. Her hydration was adequate and all other physical exam findings were normal.

No further respiratory distress was reported by the owners and her appetite was excellent. Her weight was stable and there was a very significant improvement in her overall condition. Follow-up radiographs showed that the pulmonary lesions had completely resolved with a mild pneumomediastinum secondary to the tracheotomy site. The plan was to continue tapering the prednisone dose gradually and to reexamine the patient before discontinuing the prednisone therapy.

Discussion

Treatment is usually very rewarding in cases of PIE in dogs. If possible, the inciting antigen should be eliminated. Often this is impossible to accomplish, as many times the cause for the hypersensitivity reaction is unknown. Therefore, the treatment of choice is corticosteroids.[5] Prednisone is used at an initial dose of 1-2 mg/kg per os every 12 hours.[1-4]

Clinical signs and thoracic radiographs are used to monitor response to treatment, and should be assessed weekly. Marked response to prednisone should been seen within three to five days after initiation of treatment.[3] When the clinical signs start to abate, the prednisone dose can be tapered off to the lowest effective dose over the next couple weeks to several months. Other immunosuppressive drugs such as azathioprine have been tried.[2] For acute respiratory distress, bronchodilators can be added to the supportive treatment to help with the respiratory difficulty and hypoxia.[2] The prognosis is generally fair to good, and often the response to treatment is fairly dramatic.[1,3,4]

However, lifetime treatment may be necessary for idiopathic cases.[2] Approximately 50% of these dogs will need continuous or intermittent treatment to control clinical signs.[3,5]◆

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