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Pulmonary Thromboembolism in Cats

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Pulmonary thromboembolism (PTE) is rarely diagnosed in cats, and the clinical features of the disease are not well known. PTE was diagnosed at postmortem examination in 17 cats, a prevalence of 0.06% over a 24-year period. The age of affected cats ranged from 10 months to 18 years, although young (<4 years) and old (>10 years) cats were more commonly affected than were middle-aged cats. Males and females were equally affected. The majority of cats with PTE (n = 16) had concurrent disease, which was often severe. The most common diseases identified in association with PTE were neoplasia, anemia of unidentified cause, and pancreatitis. Cats with glomerulonephritis, encephalitis, pneumonia, heart disease, and hepatic lipidosis were also represented in this study. Most cats with PTE demonstrated dyspnea and respiratory distress before death or euthanasia, but PTE was not recognized ante mortem in any cat studied. In conclusion, PTE can affect cats of any age and is associated with a variety of systemic and inflammatory disorders. It is recommended that the same clinical criteria used to increase the suspicion of PTE in dogs should also be applied to cats.

Key words: Dyspnea; Embolism; Feline; Lung; Respiratory; Thrombus.

Pulmonary thromboembolism (PTE) is a serious, and often fatal, complication of a variety of clinical disorders in humans[1] and dogs.[2] PTE in dogs is associated with immune-mediated hemolytic anemia,[3] heartworm disease,[4] hyperadrenocorticism,[5,6] and other disorders.[2,5,7] Disorders that are associated with PTE in dogs and humans often have similar pathophysiology, and PTE causes marked morbidity and mortality in both species.[1,2] A concurrent disease process was present in all the 29 cats with PTE reviewed in a retrospective study by Norris et al.[8] Although there are many reports of PTE in the dog, PTE is infrequently reported in the cat.[8–10]

In this retrospective study, we examined the case records of cats with PTE confirmed at postmortem examination to learn more about this disorder. In particular, the aims of this study were to (1) determine the prevalence of PTE in cats, (2) determine the signalment of cats that develop PTE, (3) determine the principal diseases associated with the development of PTE in cats, and (4) determine the clinical signs associated with PTE in cats.

Materials and Methods

Study Design and Inclusion Criteria

Medical records of cats with arterial pulmonary embolism confirmed at postmortem examination at Cornell University between January 1, 1975, and December 31, 1998, were examined. Affected cats were identified by a computer search of the Cornell University Hospital for Animals database with the hospital's diagnostic codes for the following conditions: PTE, lung infarction due to thrombosis, pulmonary artery thrombosis, and lung thrombosis. All cases identified by the computer search were reviewed, and cats with arterial PTE were identified.

Results

Twenty-eight cats were identified by the computer search, of which 17 were suitable for inclusion in the study. Cats identified by the computer search were excluded from the study for the following reasons: postmortem examination was not performed (n = 7), postmortem examination revealed pulmonary vein thrombus without involvement of the arterial vasculature (n = 2), clinical suspicion of PTE was not confirmed by postmortem examination (n = 1), and medical record was not available for review (n = 1). The cats affected with PTE (n = 17) were a small subset of the total number of cats (N = 30,179) examined at the Veterinary Medical Teaching Hospital over the 24-year period of this study. According to these data, the period prevalence of PTE in this population of cats was determined to be 0.06%.

Eleven of the cats with PTE were domestic shorthair cats. The remainder included the domestic long hair (n = 3), Siamese (n = 2), and Birman (n = 1) breeds. PTE affected 9 males and 8 females with nearly equal proportions. The median body weight of 15 cats with PTE was 3.8 kg (range 2.3–5.2 kg). The median age of affected cats at the time of death or euthanasia was 11 years (range 10 months–18 years). However, when plotted as a histogram, the age distribution was bimodal with peaks centered at 0 to 2 and 10 to 12 years of age (Fig 1).

PTE was associated with another disorder in 16 of 17 cats. These disorders included neoplasm (n = 6), pancreatitis (n = 2), nonhemolytic anemia (n = 2), dilated cardiomyopathy (n = 1), hepatic lipidosis (n = 1), or feline infectious peritonitis (FIP) (n = 1). Three cats with neoplasia had lymphosarcoma, 1 had metastatic adenocarcinoma, and 2 had bronchiolar carcinoma. Other disorders associated with PTE were glomerulonephritis (n = 1), bacterial pneumonia (n = 1), and encephalitis (n = 1). One cat that died from PTE had no concurrent disease detected at the time of postmortem examination.

There was an opportunity to observe for clinical signs of PTE in 16 of 17 cats. The remaining cat, which had been

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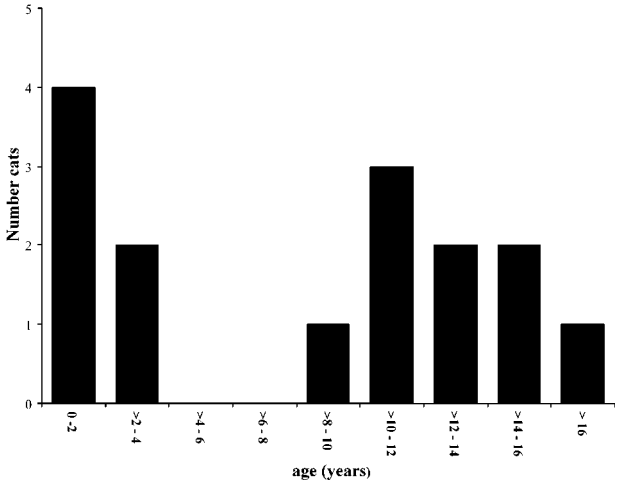

Fig 1. Age distribution of 15 cats with pulmonary thromboembolism. A bimodal distribution with peaks at 0–2 yr and 10–12 yr was noted.

diagnosed with cerebellar hypoplasia soon after birth but had been otherwise healthy, was found dead at home by the owner. Respiratory distress that immediately preceded death was recorded in 10 cats with PTE, although PTE was not suspected in any case by attending veterinarians. Clinical signs exhibited by the 10 cats with acute onset respiratory distress included tachypnea, tachycardia, increased respiratory effort, anxiousness, or open-mouth breathing. Respiratory distress in these cats was nonresponsive or only partially responsive to oxygen therapy. Seven cats died of respiratory arrest, and the remaining 10 were euthanized as a result of declining physical condition or poor prognosis. Thoracic radiographs were taken after the development of acute respiratory distress in 5 cats. Pleural effusion was the most common finding (n = 3). Other findings included alveolar opacification (n = 2), peribronchial and interstitial markings (n = 1), and pulmonary vascular congestion (n = 1).

Discussion

PTE is a rare occurrence in the cat. In this study, a computer search of more than 30,000 medical records over a 24-year period found only 17 cats with confirmed PTE. Thus, the prevalence of PTE in cats over this time period was very low (0.06%). This study was designed to exclude cats with a clinical diagnosis of PTE that was not confirmed by postmortem examination because of the difficulties associated with making an accurate antemortem diagnosis of PTE.[11] The actual prevalence of PTE in this population of cats was likely higher because some cats with small emboli might have survived and some cats that died of PTE might not have had postmortem examinations performed. On the other hand, any estimate that includes cats with only a clinical diagnosis of PTE (not confirmed with postmortem examination) is likely to overestimate the prevalence of PTE. In light of these considerations, the current data are best viewed as an estimate of the prevalence of fatal PTE in the cat.

Patient signalment was not helpful in predicting the occurrence of PTE, and the low prevalence makes it difficult to discern any breed predisposition for PTE. Most cats affected with PTE were the domestic shorthair breed, which likely reflects the overall distribution of the hospital population. The small number of purebred cats represented in this study is similar to that reported by Norris et al[8] and does not allow accurate assessment of breed susceptibility. Both sexes were equally affected in this study. The median body weight of cats with PTE in this study was 3.8 kg, similar to the body weight reported for another series of cats with PTE.[8] The low body weights of cats with PTE likely reflected the effects of concurrent illness rather than an effect of PTE. Although body-condition scores were not evaluated for the study cats, the low mean body weight likely indicates that most of these cats had less-than-optimal body-condition scores. A low body-condition score may be independently associated with an increased risk of death in emaciated cats, although age and other factors likely also play a role.[12] However, the available data do not permit us to conclude that low body weight is a risk factor for PTE. The ages of 15 of the 17 affected cats at the time of death ranged from <1 year to >18 years of age, which is similar to a previous report.[8] However, the age distribution was bimodal, with peak occurrence at less than 4 years of age or greater than 10 years of age. It is unclear why PTE occurred more frequently in younger and older cats than in middle-aged cats. One possible explanation is that disorders that place the patient at risk to develop PTE occur more often in these 2 age groups. This is true for disorders such as FIP or neoplasia, which occur more frequently in younger[13] and older cats, respectively. However, PTE also occurred in cats with pancreatitis and lymphoid neoplasms, which are disorders that affect cats of any age, suggesting that other factors might underlie the observed age distribution of affected cats.

The pathogenesis of PTE involves venous stasis, abnormalities of the vasculature endothelium, and abnormal coagulation.[5] One or more of these mechanisms are features common to the pathogenesis of many disorders and represent risk factors that promote the formation of thromboemboli. Among the disorders that have been linked to PTE in dogs are heartworm disease,[4] hemolytic anemia,[3] hyperadrenocorticism,[5] pancreatitis, neoplasia, and sepsis.[6] Dogs affected with a disorder known to be associated with PTE or with any factor or combination of factors that underlie the pathogenesis of PTE should be closely observed for signs of PTE. The results of this study and the results of others[8] indicate that most cats with PTE are also affected with severe concurrent clinical disease. Only 1 cat in the present study did not have an identifiable, concurrent medical problem. This cat had been affected with cerebellar hypoplasia since birth. In our view, cerebellar hypoplasia is unlikely to be related to PTE, so we considered this cat to be healthy for purposes of data analysis. We found that PTE complicated the clinical course of commonly diagnosed diseases of cats, such as neoplasia, pancreatitis, anemia, and cardiac disease, as in a previous study.[8] PTE also occurred in cats affected with glomerulonephritis and pneumonia, which have also been associated with PTE in dogs[5,14,15] and cats.[8] Overall, cats in the present study were affected with diseases similar to those reported in association with PTE in dogs[2,5] and humans.[1]

Only 1 study cat with confirmed PTE had heart disease, which was surprising because previous studies have reported PTE as a complication of heart disease in the cat.[8,16] To further investigate this unusual finding, we reviewed the records of cats with heart disease that had been identified by the initial computer search but were excluded from the study group. Most identified cats with heart disease had received a clinical diagnosis of PTE that was not confirmed by postmortem examination. Another possible reason so few cats with heart disease were included in our study was that these cats died outside the hospital and were unavailable for postmortem examination.

We also examined if cats with PTE manifested clinical signs of respiratory distress, if these signs were recognized as a possible indication of PTE, and if radiographic findings aided in the recognition of PTE. Ten cats with PTE were noted to have abnormal respiratory findings before death or euthanasia. The remainder of the cats either did not have an annotation in the medical record that specifically noted respiratory findings or were recorded as normal and nondistressed. Respiratory difficulty was generally ascribed by the attending clinicians to progression of an existing condition (eg, cats with known respiratory abnormalities) or to a metabolic complication of severe disease (eg, cats with pancreatitis). Interestingly, in no case studied was PTE suspected, even though the patient might have had multiple risk factors for PTE (as extrapolated from dogs and humans). Thus, we conclude that the clinical recognition of PTE in cats in this study was low.

Radiographic studies that were obtained after the onset of respiratory distress demonstrated no reliable features that might indicate PTE. The findings of pleural effusion, alveolar opacification, peribronchial and interstitial markings, or pulmonary vascular congestion were similar to those previously reported for dogs and cats with PTE.[2,8,17] In this study, radiographic abnormalities of the pulmonary vasculature were not reported in any case. It is difficult to evaluate these findings in the setting of PTE diagnosis because most of the study cats for which radiographic studies were available were those that had known thoracic disease, and the radiographs largely reflected the existing disease process. Various radiographic abnormalities of the pulmonary vasculature, including lobar oligemia, uneven or asymmetrical vascular diameter, and evidence of vessel truncation, were apparent in the majority of cats reviewed in 1 study.[8] The lack of similar radiographic findings in this study might be explained by differences in study design. In contrast with the study by Norris et al,[8] who re-evaluated the radiographic studies for each of the cats studied, we reviewed the original radiographic interpretations as reported in the medical record. Overall, the small number of radiographic studies evaluated in this study makes it difficult to form any conclusions about the usefulness of radiography in the diagnosis of PTE in cats. However, it is likely that plain radiography will have a low sensitivity and specificity for PTE in cats, as it does in dogs.[17]

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