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Pericardial Effusion in Cats: A Retrospective Study of Clinical Findings and Outcome in 146 Cats

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Background: Pericardial effusion (PE) in dogs most often is associated with neoplasia or idiopathic pericarditis, and frequently causes cardiac tamponade. Studies of PE in the cat are limited.

Hypothesis: Congestive heart failure (CHF) is the most common cause of PE in the cat.

Animals: All cats diagnosed with PE on echocardiographic examination at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania (MJR-VHUP) from 2000 to 2005.

Methods: The clinical and pathologic findings in 146 cats with PE were reviewed. Records were examined retrospectively to identify additional underlying conditions. Follow-up status and cause of death were determined by review of the medical records or phone interviews with the owners.

Results: The most common cause of PE in this study was CHF (75%). Biochemical abnormalities were uncommon, but aspartate aminotransferase (AST) activity frequently was increased (85%). Follow-up information was available on 108 cats (74%). Median survival time (MST) was 144 days for cats that were not euthanized within 24 hours (n = 85). The MST of cats with heart failure was 41 days, whereas the MST of cats without heart failure was 361 days, when those euthanized within 24 hours were excluded.

Conclusions: Survival time of cats with heart failure in this study was significantly shorter than previously reported, and significantly shorter than in cats without heart failure as a cause of PE.

Key words: Aspartate aminotransferase; Cardiomyopathy; Heart failure; Survival.

Clinically relevant pericardial disease in the cat is rare.[1,2] Previous studies have described associations between pericardial disease or effusion, or both, and concurrent feline infectious peritonitis (FIP), peritoneopericardial diaphragmatic hernia, neoplasia, cardiomyopathy, uremia, systemic infections, and idiopathic pericarditis.[1–10] Two studies have found that in cats with congestive heart failure (CHF), mild pericardial effusion (PE) was present between 46 and 49% of the time.[5,6] Other studies also have indicated that PE most commonly is associated with CHF in cats.[1,2,8] PE, especially small amounts, is a relatively common finding on echocardiographic examination of cats with cardiac disease. This finding is unlike the situation in dogs, where PE most often is associated with neoplasia or idiopathic pericarditis and frequently results in cardiac tamponade. PE as a result of CHF is uncommon in dogs and generally is small in volume when it does occur (similar to the situation in the cat).[3,7,11,12]

The purpose of the present study was to identify survival characteristics and the most common conditions associated with an antemortem diagnosis of PE in cats. Increasing numbers of general veterinary practitioners have access to echocardiography, sometimes without access to a board-certified veterinary cardiologist. Therefore, an accurate list of differential diagnoses for cats with PE is critical for devising a clinical plan for further diagnostic evaluation and treatment.

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Materials and Methods

All cardiology consultations performed on cats by clinicians in the section of cardiology at MJR-VHUP between 2000 and 2005 were searched to identify those in which PE was noted on echocardiographic examination. A total of 146 cases were retrieved from the cardiology database, with patients having more than 1 consultation only being counted once, on the earliest date that PE was recognized. Complete medical records were available for 97 cats diagnosed with PE. In cats for which the complete record was not available, additional clinical laboratory studies were accessed via the online database (available for cats seen in 2003 or later). In addition, 44 cases of cardiomyopathy were reviewed in which no PE was noted but for which serum biochemistry results were available. This comparison was done to evaluate differences in serum biochemical results between the 2 groups.

Physical examination, electrocardiographic findings, and echocardiographic examination results were recorded as interpreted by the cardiology resident or board-certified cardiologist performing the consultation. Physical examination results were considered normal if no finding was recorded in the cardiology report without a reason for the omission. Thoracic radiographs were recorded as interpreted by a board-certified radiologist or cardiologist. Additional diagnostic and hematologic information was obtained from the medical records, or the online database at MJR-VHUP. Follow-up status and cause of death was determined by review of the medical records or phone interviews with the owners.

The Kaplan-Meier product limit method was used to estimate the proportion of cats that were alive or had died. Survival time was calculated from the time of diagnosis to the date of death or last follow-up. Cats that were still alive at last follow-up were considered censored at that time. Cats that were euthanized within 24 hours due to financial concerns or poor prognosis perceived by the owner were excluded from survival analysis. Statistical differences in survival between the CHF and non-CHF cats were assessed by the log rank test. The Kruskal-Wallis test was used to compare aspartate aminotransferase (AST) between cats with PE and those with cardiomyopathy without

PE. Continuous data are presented as medians with interquartile ranges (IQR). All analyses were performed by SAS statistical software.[a] A P value $<.05$ was considered statistically significant.

Results

Signalment

Age at presentation ranged from 7 months to 23 years. There were 93 neutered males, 45 spayed females, 7 intact females, and 1 intact male. Breed distribution was as follows: 118 domestic short hair, 12 domestic long hair, 5 Maine Coon, 4 Persian, 3 Himalayan, 2 Siamese, 1 Norwegian Forest Cat, and 1 Birman. Seventy-eight percent were indoor only cats.

Physical Examination

Complete physical examination findings were not recorded on 6 cardiology reports because of overly fractious nature (2), loud vocalizing (1), muffling due to severe pleural effusion (1), being under anesthesia (1), and highly critical condition progressing to death shortly after completion of the echocardiographic examination (1). Additional findings that were not recorded on other cardiology reports and assumed to be normal included respiratory rate (22), lung sounds (18), presence or absence of a heart murmur (9) or gallop sound (19), heart rate (4) or rhythm (4), pulse quality (14), and body temperature (64). Abnormalities noted on physical examination included tachypnea (83/140), abnormal lung sounds (68/140), heart murmur (65/140), gallop rhythm (53/140), decreased pulse quality (38/140), arrhythmia (22/140), hypothermia (30/76), bradycardia (9/140), tachycardia (6/140), and hyperthermia (3/76).

Electrocardiography

Electrocardiograms (ECGs) were recorded in 126 cats. Rhythm abnormalities included premature ventricular complexes (16%) and atrial fibrillation (3.9%). Conduction abnormalities included 6 cats with left bundle branch block (4.7%), with 5 of these recorded as having left anterior fascicular block. Other conduction abnormalities included right bundle branch block (2.3%), complete atrioventricular (AV) block (2.3%), first degree AV block (1.6%), and ventricular preexcitation (1 cat). The mean electrical axis was shifted in approximately 20% of the cats (left axis deviation 11.1%; right axis deviation 8.3%). Four cats (5.4%) had small QRS complexes.

Echocardiography

Standard echocardiographic measurements were recorded in all cats by the recommendations of the American Society of Echocardiography, including the left ventricular internal diameter (LVID) during diastole and systole, the diastolic interventricular septal thickness (IVS), and the diastolic posterior wall thickness (PW). The fractional shortening was calculated by the following formula: (LVID diastole − LVID systole) ÷ LVID diastole. All measurements were obtained from an M-mode echocardiographic examination. The aorta and left atrial diameter were measured on the short-axis view obtained from the right parasternal window as previously described.[13]

Hypertrophic cardiomyopathy (HCM) was the most commonly diagnosed disease (n = 39), followed by unclassified cardiomyopathy (UCM, n = 31) and restrictive cardiomyopathy (RCM, n = 19). Dilated cardiomyopathy (DCM) was less commonly found (n = 4). Only 3 cats had PE classified as more than mild. Other cardiac abnormalities or diseases with cardiac implications included cardiac neoplasia (7), arrhythmogenic right ventricular cardiomyopathy (3), thyrotoxicosis (2), primary mitral valve disease (2), pulmonary hypertension (1), aortic stenosis and mitral valve dysplasia (1), and pericardial peritoneal diaphragmatic hernia (1).

Thoracic Radiography

Cardiomegaly frequently was noted (95%). Pleural effusion (79%) and abnormal pulmonary patterns (60%) were relatively common findings. A thoracic mass was noted in 5 cats.

Clinical Laboratory Studies

Hematologic and serum biochemical determinations included pH, white blood cell count, platelet count, hematocrit, total solids, prothrombin time, partial thromboplastin time, glucose, blood urea nitrogen (BUN), creatinine, phosphorus, calcium, sodium, potassium, chloride, carbon dioxide, total protein, albumin, globulin, alanine aminotransferase (ALT), AST, alkaline phosphatase, gamma glutamyl transferase (GGT), total bilirubin, cholesterol, anion gap, magnesium, urine specific gravity, and l-thyroxine). Results were considered abnormal if outside the laboratory reference range, except for prothrombin time and partial thromboplastin time, which were considered abnormal if >25% above the upper range of normal. Serum biochemical results generally were normal. AST activity was the most frequently increased biochemical result (85%), with a median value of 55 U/L (IQR, 40–94.5). In 44 cats with cardiomyopathy but without PE, AST activity also was frequently increased (82%), with a median value of 53.5 U/L. There was no significant difference between these groups (P = 0.97). Of the cats with PE but without cardiac disease, 63% (7/11) had increased AST activity. These cats had significantly lower AST activity than did either cats with cardiomyopathy or cats with PE and cardiac disease (median, 41 U/L; P = .04).

Clinical Pathology Results

Forty-three cats had 1 or more cavitary effusions submitted for cytology. Pleural effusion samples were most commonly submitted, with chylous effusion, modified transudate, and transudate being the most

Table 1. Assumed cause of pericardial effusion.

Assumed Cause of Pericardial Effusion	Number	Total %
Congestive heart failure (other causes ruled out)	81	55.5
Congestive heart failure versus other	15	10.3
Congestive heart failure/fluid overload	14	9.6
Open	12	8.2
Neoplasia	8	5.4
Idiopathic	6	4.1
Uremia versus fluid overload	5	3.4
Thyrotoxic cardiomyopathy	3	2.1
Idiopathic with FIP suspected	1	0.7
Pericardioperitoneal diaphragmatic hernia	1	0.7
Total	146	100

FIP, feline infectious peritonitis.

common diagnoses. Two cats had pericardial fluid submitted. One sample was chylous and the second was an exudate with mesothelial hyperplasia and dysplasia.

Assumed Cause of Pericardial Effusion

Assigning the cause of PE was based on the echocardiographic assessment by the cardiologist performing the examination, and concurrent diagnostic findings. In some cases, a primary cause could not be identified, or more than 1 underlying etiology was thought to be present (Table 1).

Diagnoses

Of 146 cats, 129 (88.3%) had primary cardiac disease and 102 (69.9%) had 1 or more noncardiac diseases present. Of the cats with primary cardiac disease, 19 had a rhythm or conduction abnormality but were otherwise normal on echocardiographic examination. Forty-three (29.4%) were azotemic, with 17 (40% of the azotemic cats) having renal azotemia; 25 (17.1%) had extracardiac neoplasia; 7 (4.8%) had cardiac neoplasia; and 61 (41.8%) had another concurrent disease. Anemia was the most common underlying abnormality, with 38 (26%) cats affected; 10 of these cats had nonregenerative anemia.

Of the cats with primary cardiac disease, 85 (66%) had an additional disease present. Thirty-nine (30%) of these cats had azotemia, with 14 (35.9% of azotemic cases) having renal azotemia; 17 (13%) had extracardiac neoplasia; 3 (2.3%) had cardiac neoplasia and 52 (40%) had another underlying disease.

The majority of cats with extracardiac diseases also had primary cardiac disease. Of the 43 cats with azotemia, only 4 (9.3%) did not have cardiac disease, and 3 of these 4 cats had chronic renal failure. Of the 61 cats with concurrent disease, 9 (14.7%) did not have cardiac disease. Of the 25 cats with extracardiac neoplasia, 8 (32%) did not have cardiac disease. Of the 7 cats with cardiac neoplasia, 4 (57%) did not have other cardiac disease.

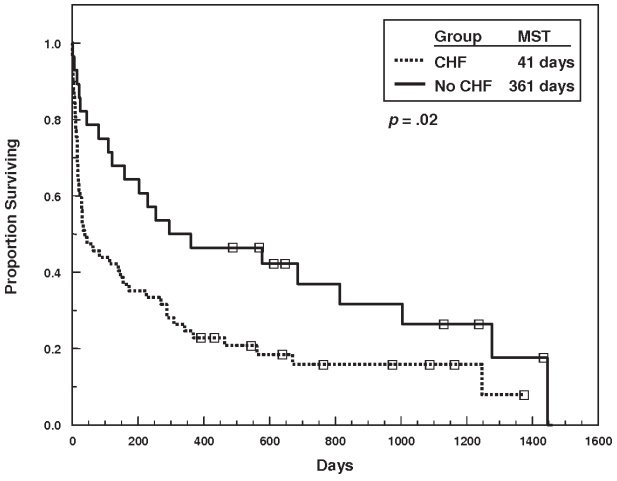

Fig 1. Survival curve of 85 cats according to presence vs absence of CHF at time of initial diagnosis of PE.

Survival Analysis

Follow-up was available on 108 cats (74%). Median survival time (MST) was 144 days for cats that were not euthanized within 24 hours (n = 85). Cats without heart failure had significantly longer survival compared with cats with heart failure (MST, 361 days for cats without CHF and 41 days for cats with CHF; $P = .02$; Fig 1).

Discussion

The most common cause of PE in this study was CHF (75% prevalence) with HCM being the most common underlying cardiac disease (n = 39), followed by UCM (n = 31) and RCM (n = 19). A previous retrospective study evaluated pericardial disease in 66 cats at postmortem examination[2] and found FIP most commonly associated with pericardial pathology (17%). CHF (secondary to HCM, DCM, RCM, or mitral valve disease) was associated with only 28% of the cats. This difference likely is related to the inherent differences in study populations. The cats in this report presented to the cardiology service for evaluation of suspected cardiac disease, presumably leading to underrepresentation of other systemic diseases in which PE may have been present but not suspected on physical examination. Furthermore, we did not conduct a postmortem

examination–based search for pericardial disease or effusion or both, which likely led to underrepresentation of neoplasia, FIP, and other systemic diseases that can lead to pericardial pathology. Finally, it is possible that small amounts of PE were not detectable on postmortem examination leading to another potential cause of discrepant findings.

In contrast to clinically relevant volumes of PE that may be seen with diseases such as FIP and neoplasia, the volume of PE seen in cats with CHF is often mild and secondary to passive congestion.[1,5,6,8] Liu reported an average volume of 5.9 mL (range, 2.0–25 mL) of pericardial fluid removed from 112 cats identified on postmortem examination as having died of heart failure.[5] To the authors' knowledge, only 1 case has been reported in which PE associated with CHF in a cat was sufficient to cause clinical signs.[4] The cat in that report experienced marked clinical improvement after 50 mL of hemorrhagic fluid was removed by pericardiocentesis. A partial pericardiectomy then was performed. The cat was treated medically for congestive cardiomyopathy and was clinically compensated at follow-up 1 year later. The reported cat also had pericarditis, which was assumed to be secondary to fibrosis induced by chronic pericardial stretching, but a viral etiology could not be ruled out. Pericardial fluid was only obtained from 2 cats in this study: 1 sample was chylous and associated with CHF; an inflammatory exudate was found in the other cat. The latter cat was diagnosed with a mesothelioma and recovered after pericardiectomy. In the cat with CHF (severe HCM), 50 mL of chylous fluid was removed from the pericardial space, and 210 mL was removed from the pleural space on emergency before being evaluated by the cardiology service. It is unclear whether or not cardiac tamponade was identified before fluid removal. This cat also was diagnosed with chronic renal failure, and PE may have been multifactorial. In dogs, PE is an uncommon finding secondary to CHF and occurs rarely with left atrial rupture.[3,7,11,12]

Thirty two (21.9%) of the cats in this study had neoplasia. Neoplasia was determined to be the sole cause of PE in 8 (5.5%) cats and may have been a contributing factor in an additional 11 (7.5%) cats. Seven (4.8%) cats had masses identified on echocardiography, a lower percentage than has been reported in dogs. In 1 study of 143 dogs with PE, a mass was identified on echocardiography in 44 dogs (30.8%).[12] These results support the hypothesis that neoplasia is a less frequent cause of PE in cats than in dogs. Rush et al reported 12 cases (18%) of metastatic neoplasia as the cause of PE in an analysis of 66 cats examined postmortem.[2] Although a higher percentage than we report, it is difficult to compare these studies, as discussed previously. The most commonly reported type of neoplasia in this study was lymphosarcoma (n = 10), which is in agreement with previous studies reporting lymphosarcoma as the most common neoplasm associated with pericardial pathology in the cat.[2,3,7,8] In addition, 4 cats were diagnosed with pulmonary carcinoma; 1 cat with abdominal carcinoma; 1 cat with fibrosarcoma; 1 cat with mesothelioma; and in 15 cats a histologic or cytologic diagnosis was not obtained, including 5 cats with suspected abdominal carcinoma.

Ninety-two of the cats in the present study had serum AST activity measured, with a median value of 55 U/L (IQR, 40–94.5). Seventy-eight (85%) of these cats had increased serum AST activity. To investigate whether this finding was specific to cats with PE, we evaluated a separate group of 44 cats diagnosed with cardiomyopathy in which no PE was noted. These cases were obtained from the online database at MJR-VHUP by use of a diagnosis of cardiomyopathy, lack of PE, and biochemistry profile results (including AST) as inclusion criteria. In this group, AST also was frequently increased (82%), with a median value of 53.5 U/L. There was no significant difference between these 2 groups ($P = .97$), suggesting that increased AST activity is not specific for cats with PE. In 11 of the cats with PE in which no cardiac disease was found, 7 (63%) had increased AST activity, with a median value of 41 U/L. These cats had significantly lower AST activity than did either the 44 cats with cardiomyopathy or the remaining 81 cats with PE and cardiac disease ($P = .04$). Although increased AST activity may be seen with hepatic disease or myopathy, previous studies examining serum biochemical findings in cats with various diseases have identified increased AST activity associated with anorexia, hyperthyroidism, and arterial thromboembolism.[14–17] Based on the small number of cats in this study, and some overlap in AST activity between cats with and without cardiac disease, it is difficult to conclude whether mild to moderate increases in AST activity may also be a biochemical marker for cardiomyopathy in the cat, although additional studies may answer this question.

Uremia has previously been associated with PE in the dog and cat.[1–3,7,11] Possible mechanisms for development of PE in the uremic state include damage of the capillary endothelium producing epicardial serositis or hemorrhage secondary to impaired platelet function.[2] Of the 43 cats with azotemia in this study, only 17 (39.5%) had primary renal disease. Many of the cats with preexisting CHF were treated with large doses of diuretics with subsequent development of mild to moderate azotemia at the time of echocardiographic examination that later resolved. In these cats with preexisting CHF, uremia was not considered as a possible cause of PE. Determining the underlying cause of PE in cats with both renal disease and primary cardiac disease can be difficult, and both potentially may contribute. In this study, 17 (11.6%) or fewer cats were considered to have some degree of PE caused by uremia. Another commonly recognized finding in renal failure is systemic hypertension, which can contribute to ventricular hypertrophy, further complicating the diagnosis of HCM versus secondary hypertrophy. These confounding factors make it difficult to ascertain a primary cause of PE when multiple disease processes are present. In this study, however, when a noncardiac disease was present, primary cardiac disease also was present in most instances. For example, of 61 cats with other concurrent

disease (excluding azotemia and neoplasia), only 9 (14.7%) did not have cardiac disease. In contrast, of 129 cats with primary cardiac disease, 44 (34.1%) did not have any additional diseases present. Although this association may be partly because of the population studied (ie, cats with clinical signs suggesting heart disease and prompting a cardiology consultation), cardiomyopathy and CHF should be ruled out first when evaluating cats with PE. The amount of PE noted on echocardiographic examination in this study usually was mild, making it difficult to make associations between the volume of PE and disease state.

Physical examination, radiographic findings, and electrocardiographic findings were similar to findings in cats with cardiomyopathy. Electrical alterans was not reported in any cats in this study despite 3 cats having PE classified as moderate to severe. In the cat that had 50 mL of chylous effusion removed from its pericardial space, the ECG rhythm diagnosis was sinus tachycardia with polymorphic ventricular premature contractions. In the 2 other cats with moderate to severe PE, ECGs were not recorded. Given that the remainder of cats in this study had mild amounts of PE without evidence of hemodynamic complications, we would not expect to find clinical signs associated with cardiac tamponade.

Previous studies of cardiomyopathy in cats have demonstrated substantially longer survival times than we found in this population of cats. Ferasin et al reported the MST of 73 cats with various forms of cardiomyopathy to be 300 days. Cats with UCM lived longer (925 days) than those with HCM (492 days), RCM (132 days), or DCM (11 days).[18] Rush et al reviewed 260 cases of HCM, in which overall MST was 709 days (range, 2–4,418 days) for cats that survived greater than 24 hours. Lower survival times were reported for cats in the CHF group (563 days) and cats in the arterial thromboembolism group (184 days).[19] Overall MST from the first diagnosis of PE in this study was 144 days for cats not euthanized within 24 hours, with an MST of 41 days for cats with CHF. We excluded cats euthanized within 24 hours due to financial concerns or perceived poor prognosis by the owner, because survival time in these cats was decreased by factors not related to their disease. Cats with PE in association with CHF appear to have a more guarded prognosis than cats with PE unrelated to CHF. However, 70% of the cats in this study had 1 or more noncardiac diseases present, including 32 cats with cardiac or noncardiac neoplasia, suggesting that these cats may have been sicker than those evaluated in the previous studies. Finally, survival times reported in this study were from the initial diagnosis of PE, not from the time that cardiomyopathy or CHF was first diagnosed.

Several limitations are inherent in the design of this study. Complete medical records were available for only 97 of the cats evaluated, leading to potential omission of clinical findings, history, and laboratory results for cats evaluated before 2003 (ie, before clinical laboratory results were entered into the online database at MJR-VHUP). We did not include a postmortem examination–based search of cats with PE in this study, thus leading to underrepresentation of noncardiac causes of PE. In addition, the presence of more than 1 underlying disease process posed difficulties in defining a primary cause for PE in some cats. Survival analysis for heart disease alone is not possible from this study because survival was measured from the time PE was first recognized, not necessarily from the time cardiomyopathy or CHF or both were initially diagnosed. Additional studies would be required to evaluate whether a significant difference in survival characteristics exists between cardiomyopathic cats with and without PE.

Conclusion

The most common cause of PE in this study was CHF, and the presence of PE on echocardiographic examination may be useful as a marker defining progression of CHF or response to therapy or both. Although the survival times reported here were substantially shorter than previously reported for cats with cardiomyopathy, additional studies are needed to compare survival times in cats with CHF presenting with and without PE.

Footnotes

[a] version 9.1, SAS Institute, Cary, NC

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