Idiopathic Chylothorax and Fibrosing Pleuritis in a Cat

INTRODUCTION

This case report describes an eight-year-old neutered male domestic longhaired cat diagnosed with idiopathic chylothorax and fibrosing pleuritis. A diagnosis of idiopathic chylothorax was made by fluid analysis and exclusion of other known causes of chylothorax. The case was medically managed with intermittent thoracocentesis, chest tube placement, a low fat diet, rutin, corticosteroids, and somatostatin. The cat experienced a remission of clinical signs for seven months, then relapsed and was euthanized ten months after initial diagnosis. Necropsy was declined by the owner.

Chylothorax is an uncommon, highly fatal disease in cats.[1] This condition is characterized by the accumulation of chyle, a liquid that is composed of lymphatic fluid containing a high quantity of fat and protein derived principally from the intestine, in the pleural space. Chyle is normally transported to the venous system via a network of lymphatics in the mesentery. These lymphatics feed into the cisterna chyli, which lies adjacent to the aorta at L1-L4. Cranially, the thoracic duct continues from the cisterna chyli and runs between the aorta and the azygous vein on the left side of the thorax and empties at the junction of the left jugular vein and the brachiocephalic vein[2]. The basal rate of lymph flow in dogs is 2 ml/kg/hr and is assumed to be similar in cats[2,3]. The rate of chylous flow is greatest after a high-fat meal. Seventy percent of all ingested fats are conveyed to the blood stream by the thoracic duct; protein is also transported to the venous system from the capillary spaces via the thoracic duct.[2] Therefore, loss of chyle into the thorax can result in respiratory distress, loss of lymphocytes, and malnutrition due to the loss of protein, fat, and fat-soluble vitamins.

In humans, chylothorax is most commonly associated with trauma to the thoracic duct (usually during thoracic surgery), neoplasia, or congenital abnormalities.[4] In contrast, chylothorax in cats is rarely due to trauma; the thoracic duct has been shown to heal rapidly after injury, with the effusion resolving without

surgical treatment in one to two weeks.[5] In cats, chyle leaks from intact, dilated thoracic lymphatic vessels (thoracic lymphangiectasia) and is thought to result from abnormal pressures or flows within the thoracic duct.[6] Increased lymphatic flow can result from greater hepatic lymph formation. Decreased lymphatic drainage into the venous system can result from increased venous pressure. Known causes of chylothorax include anterior mediastinal masses (mediastinal lymphoma, thymoma), heart disease (cardiomyopathy, pericardial effusion, congenital heart disease), venous thrombi, heartworm disease, fungal disease, feline infectious peritonitis, and congenital defects of the thoracic duct.[5] Other etiologies include trauma, pulmonary neoplasia, diaphragmatic hernia, peritoneopericardial hernia, lung lobe torsion, and hyperthyroidism with resulting cardiomyopathy.[6,7] In the majority of cats (>90%), the cause of chylothorax is not elucidated despite extensive work-up and is thus deemed idiopathic.[2] However, every effort should be made to determine the etiology of the disease and any underlying conditions as treatment varies depending on the cause of chylothorax. One theory postulates that animals with idiopathic chylothorax have increased volumes of lymph being transported through the thoracic duct. These increased flows may occur secondary to increased right-sided venous pressures that cause lymph that would normally be transported from the liver to the venous system to be shunted into the lymphatic system. Minimally elevated venous pressures, in association with other unknown factors, could substantially decrease lymphatic drainage into the venous system.[8]

Fibrosing pleuritis can result from any prolonged exudative or hemorrhagic condition in the thorax.[9] Fibrosing pleuritis is characterized by pleura thickened by diffuse fibrous tissue which restricts normal pulmonary expansion. Effusions are irritating to the pleural surface, and chronic pleural irritation from chylous fluid contributes to fibrosing pleuritis.[9] Radiographic documentation of irregular or rounded lung lobe edges and persistent lung lobe collapse despite effective thoracic drainage supports a diagnosis of restrictive pleuritis[10]. Ultrasound may show fluid confined by fibrinous adhesions and fluid distribution that is not affected by gravity. Fibrin tags may be seen floating in the fluid. Cats with fibrosing pleuritis may by misdiagnosed as having large amounts of pleural effusion, when in fact pleural effusion is minimal.[9] Ultrasound helps to differentiate the conditions. Fibrosing pleuritis should be suspected in cats with persistent dyspnea and minimal pleural fluid. Up to sixty-three percent of cats with chylothorax

develop fibrosing pleuritis[10]. Severe fibrosing pleuritis has a grave prognosis and cats with this condition are less likely to respond to medical or surgical treatment of chylothorax.[10, 11]

Chylothorax most commonly manifests in middle-aged cats, with a median age of eight years; however, cases have been reported in cats ranging from six months to fifteen years of age. No sex predisposition has been reported.[1] Siamese and Himalayan cats have an increased prevalence.[5] The most common presenting complaints are lethargy, decreased activity, dyspnea, and cough. Often the duration of clinical signs is greater than one month, suggesting slow accumulation of fluid in the pleural space. Coughing may result from pleural irritation. Ventilatory efforts are characterized by a restrictive pattern (increased frequency of respiration with prolonged inspiration and delayed expiration). A history of anorexia or decreased appetite is common. Vomiting or regurgitation can be associated with the presence of a mediastinal mass. Physical examination often reveals muffled heart sounds, increased bronchovesicular sounds at the dorsal aspect of the thorax with absent lung sounds ventrally. Weight loss is common. Most cats with chylothorax present normo-thermic; pyothorax should be suspected in cats with elevated temperatures, although bacterial infections secondary to thoracocentesis or surgery can occur. Other findings may include tachycardia, cardiac murmurs, arrhythmias, ascites, depression, and pale mucous membranes. Decreased chest compressibility suggests a mediastinal mass. Diagnostics should be performed with care – most cats with chylothorax have a large amount of fluid accumulation before symptoms occur and cats may decompensate rapidly with the stress of examination or diagnostics. In many cases, complete diagnostics should be delayed until the cat is stabilized with thoracocentesis and oxygen therapy. If the cat is not overtly dyspneic, thoracic radiographs should be taken to confirm the presence of pleural fluid. A dorsoventral view usually compromises respiration less than a ventrodorsal view. Pleural effusion is characterized radiographically by pleural fissure lines, rounding of lung borders, mediastinal widening, separation of lung margins from the thoracic wall, and an obscure cardiac silhouette. Alternatively, ultrasound can be used in a minimally restrained cat to confirm the presence of fluid and guide thoracocentesis.

Thoracocentesis and fluid analysis are important components of diagnosing the cause of pleural fluid accumulation. Thoracocentesis should be performed between the seventh and ninth intercostal spaces in

the ventral half of the thorax, avoiding the intercostal vessels caudal to the ribs. A 21 gauge butterfly catheter or a 22 gauge over-the-needle catheter should be used. The author prefers an over-the-needle catheter as the stylet can be removed once the catheter is placed, decreasing the chance of lung laceration if the patient moves during aspiration. Pleural effusion can be formed by any of the following mechanisms: increased venous hydrostatic pressure, decreased capillary oncotic pressure, increased capillary membrane permeability, lymphatic obstruction or leakage, or hemorrhage. Pleural fluid should be categorized into one of the following types: hemorrhage, chylous effusion, septic exudate, nonseptic exudate, transudate, and modified transudate. Chylous fluid is a white or pinkish opaque fluid in which a cream layer will form when left to stand. Chyle will not separate when centrifuged. The protein content of chyle is between 2.5 and 4.0 gm/dl, and the total nucleated cell count is usually between 1000 and 7000 cells/microliter.[1] The predominant cell type consists of small lymphocytes with lesser numbers of lipid-containing macrophages. With increased chronicity, nondegenerative neutrophils may predominate indicating increased inflammation. Degenerative neutrophils and bacteria are uncommon due to the bacteriostatic effect of lecithin and fatty acids, though infection may be introduced iatrogenically by thoracocentesis.[2] Aerobic and anaerobic culture of thoracic fluid should always be performed. To confirm the presence of chylous effusion, a comparison of serum and thoracic fluid triglyceride levels should be performed.[12] True chylous effusions have a higher triglyceride concentration than serum. A fluid cholesterol to triglyceride ratio <1 is supportive of a diagnosis of chylous effusion.[6] Other tests that can aid in confirming the presence of a chylous effusion include Sudan III stain for lipid droplets and the ether clearance test. Both tests are crude estimates of fat content. However, the absence of fat droplets does not rule out chylothorax.[12]

Once the presence of chylous fluid is confirmed, it is imperative to perform further diagnostic tests to attempt to determine etiology as treatment should be geared at the underlying problem whenever possible. Thoracic ultrasound can evaluate cardiac structure and function, valvular lesions and function, presence of pericardial effusion, presence of masses or thrombi occluding the vena cava, mediastinal masses, and diaphragmatic integrity/presence of diaphragmatic hernia. If a mediastinal mass is identified, a fine needle aspirate should be performed to determine the tissue type. Heartworms may be visualized in the pulmonary outflow tract. Ultrasound is best performed before large quantities of fluid are removed, as the fluid acts as

an acoustic window that enhances visualization of the thoracic structures. Fibrinous pleuritis is suspected when fluid is confined by fibrinous adhesions and fluid distribution is not affected by gravity. Fibrin tags may be seen floating in the fluid.

Radiographs should be repeated after fluid removal and stabilization of the patient. Radiographs should be assessed for evidence of mediastinal masses, rib fractures, mass lesions of the pulmonary parenchyma, or diaphragmatic hernia. Elevation of the trachea or persistent soft tissue density in the cranial mediastinum is suggestive of a mediastinal mass. Failure of lung lobes to re-expand after removal of pleural fluid and rounded or irregular lung borders increases suspicion of fibrinous pleuritis. Atelectic lung lobes can be confused with pulmonary neoplasia, lung lobe torsion, or hilar lymphadenopathy. Where available, computed tomography is helpful in differentiating pleural fibrosis from neoplasia or other abnormalities if the patient is stable enough to undergo anesthesia.[2]

A complete blood count (CBC) and biochemistry analysis is needed in all cats diagnosed with chylothorax to detect underlying or concurrent disease. A serum T4 and free T4 by equilibrium dialysis (fT4 by ED) should be included to rule out hyperthyroidism. Serum heartworm antibody and antigen levels should be performed. Feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) status should be assessed to aid in predicting prognosis.

Although ninety percent of cases of chylothorax are idiopathic, whenever possible therapy of chylothorax should be directed at the underlying cause. Treatment of chylothorax varies considerably depending on the underlying cause, and thorough work-up is critical in developing an appropriate treatment plan. Cats with hyperthyroidism should be treated with radioactive iodine, methimazole, or surgery. Underlying cardiac disease must be treated with appropriate medical management. Surgery for thymoma relieves compression of the cranial vena cava and may resolve chylothorax. If lymphoma is present, chemotherapy may alleviate chylothorax. During treatment of any underlying cause of chylothorax, dyspnea related to chyle accumulation should be controlled with periodic thoracocentesis. A chest tube should be placed if thoracocentesis to control dyspnea is required more often than once weekly. Most cats have an incomplete

mediastinum, allowing adequate drainage from a unilateral chest tap or chest tube. However, some cats have a complete mediastinum or develop fibrinous occlusion of the mediastinum, necessitating bilateral drainage.

Medical management of idiopathic chylothorax consists of periodic evacuation of the thorax combined with medical measures designed to decrease the formation of chyle. A low-fat diet to decrease chyle flow has been advocated in treating idiopathic chylothorax. In normal dogs and humans, however, changes in dietary fat content has not been shown to significantly alter thoracic duct flow[5]. Similar studies have not been performed in cats. Supplementing the diet with low- or medium-chain triglycerides has been recommended because these fats were thought to be absorbed directly into the portal system, bypassing the thoracic duct. It has since been discovered, however, that these fatty acids do increase thoracic duct flow in dogs and humans, and their use is no longer recommended.[5]

Rutin, a bioflavinoid, has been advocated for the treatment of chylothorax. Bioflavinoids have several possible modes of action, including increased protein removal from lymphatic vessels, decreased blood vessel leakage, induction of phagocytosis via macrophage stimulation, and increased proteolysis and lymph removal from tissues.[13] No published studies have proven the effectiveness of rutin.

Octreotide, a synthetic somatostatin, has been used successfully in the treatment of chylothorax in humans. Somatostatin decreases intestinal absorption of fats and triglyceride concentrations in thoracic duct lymph.[14] It is unknown whether somatostatin directly interferes with triglyceride transport in the gut wall, or if triglyceride delivery to the thoracic duct is reduced by decreasing intestinal blood flow or motility[14]. Octreotide markedly inhibits basal and pentagastrin-stimulated gastric acid secretion, blocks pancreatic secretion, and inhibits biliary secretion. As a result of the decrease in GI secretory volume and enzymes, the amount and protein content of the fluid in the thoracic duct is reduced.[15] Octreotide has been shown to be effective in the treatment of experimentally-induced chylothorax in dogs.[16] Currently a study is underway at the University of Wisconsin Madison School of Veterinary Medicine to evaluate the efficacy and safety of octreotide in naturally-occurring chylothorax in cats.

Surgical intervention may be warranted in cats in which medical management fails or becomes impractical, such as in cats that require thoracocentesis more frequently than once weekly. Some authors believe that surgical intervention is warranted early in the course of the disease to prevent fibrosing pleuritis.[2,6,7,8] Surgical options include thoracic duct ligation, subtotal pericardiectomy, passive pleuroperitoneal shunting, active pleuroperitoneal or pleurovenous shunting, pleurodesis, and omentalization of the thorax. Thoracic duct ligation is the most frequently performed procedure. No long-term studies compare the success of thoracic duct ligation and other techniques, and most studies describe very small numbers of cases (one to three cats). Therefore, it is difficult to confidently recommend one technique over another. The choice of surgery type often is dependent on the experience and preference of the surgeon. Theoretically, techniques such as thoracic duct ligation that decrease chyle leakage into the thorax should have greater efficacy in preventing fibrosing pleuritis.

Thoracic duct ligation is thought to cause abdominal lymphatico-venous anastomoses to form, causing chyle to bypass the thoracic duct.[8] The reported success rate of this procedure varies from twenty to sixty percent.[1,6,17,18] Ligation is performed via a left lateral intercostal thoracotomy or transdiaphragmatically. Advantages of successful thoracic duct ligation include complete resolution of chylothorax and prevention of fibrosing pleuritis. The disadvantages include a long operative time, high incidence of failure, and technical difficulty in the cat.[17] Fifty-six percent of cats survived more than twelve months after surgery in one study. [18] Pericardiectomy may be performed in conjunction with thoracic duct ligation or as a sole procedure. Thickening of the pericardium occurs in many cats affected with chylothorax. The thickened pericardium causes an elevation in venous pressures, which can result in formation of numerous lymphatics in the cranial thorax.[8]

Successful treatment of chylothorax by advancement of the omentum into the thorax was described in one cat.[19] A thoracotomy is made at the fifth or sixth intercostal space. A paracostal incision is made to raise a dorsal omental pedicle flap, which is then brought through an incision in the pars costalis of the diaphragm. The omentum is tacked via omentopexy to the mediastinum between the thoracic duct and the cranial vena

cava. [19] The true function of omentum in the treatment of chylothorax is unknown. The omentum contains aggregates of lymphoid tissue that provide efficient lymphatic drainage. These lymphatics eventually drain to the subpyloric and splenic lymph nodes, which then drains back into the thoracic duct and eventually drains back into the thoracic cavity. Therefore, omentum may aid in resolving chylothorax via the angiogenic and adhesion-forming properties of the omentum rather than by direct physiologic drainage. [19]

Passive pleuroperitoneal shunting via a fenestrated silastic sheet in the diaphragm allows chyle to drain into the abdomen. Chyle is absorbed by the omental lymphatics and is eventually drained back into the thoracic cavity. Chronic irritation induced by the sheeting has been associated with neoplastic transformation of tissues.[8] Active pleuroperitoneal or pleurovenous shunting has also been used in the treatment of chylothorax. Active shunting may provide more complete drainage of the thorax than passive shunting.[8] However, shunts can occlude with fibrin and they require a high degree of owner compliance. Thrombosis, venous occlusion, sepsis, and electrolyte abnormalities have been reported in humans treated with pleuroperitoneal and pleurovenous shunts. [8]

Pleurodesis is the induction of generalized adhesions between the visceral and parietal pleura by infusing irritating substances into the pleural cavity. In order for pleurodesis to occur, the lungs must be able to contact the body wall, which may be impossible in the presence of fibrosing pleuritis. Surgical pleurodesis and talc administration both failed to induce pleurodesis in experimental dogs.[20] Pleurodesis with tetracycline hydrochloride has been described, but is rarely successful in treating chylothorax.[1,7] Additionally, pleurodesis is an extremely painful procedure; this technique is not recommended in the treatment of chylothorax in cats.

The prognosis for feline chylothorax has not been evaluated in a large number of cats. Generally, the prognosis for chylothorax is guarded, with most cats surviving a few months to a year or more[1,5,8,10, 18]. Many cats are euthanized due to the need for repeated thoracocentesis, severe dyspnea, emaciation, or cost of treatment.

CLINICAL REPORT

An eight-year-old neutered male 5.4 kg Domestic longhaired cat was presented for a mildly increased respiratory rate for one day. His appetite and activity were normal, and he had no history of vomiting, diarrhea, or cough. The cat was an indoor/outdoor cat, and had current vaccinations for feline calicivirus, feline herpesvirus, panleukopenia, rabies, and feline leukemia. On presentation the cat was bright and alert with a slightly increased respiratory rate of 46 breaths per minute (normal 20-30 breaths per minute). Heart sounds were muffled bilaterally, no murmurs were asculted, and breath sounds were absent ventrally and normal dorsally. Heart rate was normal at 170 beats per minute. Body temperature was 38.0°C (normal 38.0 to 39.5°C). The abdomen palpated normally. Mucous membranes were pink and moist with a capillary refill time of one second. The cat was judged stable enough for radiography, and thoracic radiographs revealed moderate bilateral pleural effusion (Figure 1). No mass lesions were evident in the lung fields, no rib fractures were visible, and the cardiac silhouette appeared normal, although complete evaluation was not possible due to the volume of effusion. Differential diagnoses for pleural effusion were hemorrhage (trauma, coagulopathy), exudate (infection/pyothorax), transudate (hypoproteinemia), modified transudate (neoplasia, congestive heart failure, caval syndrome, fungal infection, pancreatitis, FIP), and chylothorax.

Thoracocentesis was performed with a 22 gauge over the needle catheter on the right side at the eighth intercostal space. One hundred fifty milliliters of milky white opaque fluid was recovered. The cat's respiratory rate decreased to 20 breaths per minute following thoracocentesis. Samples of fluid were placed in lavender-top (EDTA) tubes for submission for cytology and fluid analysis and in red top tubes for culture. Centrifugation failed to clear the fluid. Rapid on-site cytological evaluation was made by placing a drop of fluid on a slide and adding a drop of Wright's stain. Crenated red blood cells, small well-differentiated lymphocytes, and a small number of lipid-laden macrophages were seen. A tentative diagnosis of chylothorax was made pending results of fluid and serum triglyceride levels. Differential diagnoses included anterior mediastinal masses (mediastinal lymphoma, thymoma), heart disease

FIGURE 1: RADIOGRAPHS OF PLEURAL EFFUSION PRIOR TO THORACOCENTESIS

(cardiomyopathy, pericardial effusion, congenital heart disease), venous thrombi, heartworm disease, fungal disease, feline infectious peritonitis, trauma, pulmonary neoplasia, diaphragmatic hernia, peritoneopericardial hernia, lung lobe torsion, hyperthyroidism with resulting cardiomyopathy, and idiopathic chylothorax.

Radiographs were repeated after thoracocentesis and showed nearly complete drainage of the thorax with re-expansion of lung lobes. No evidence of neoplasia, mediastinal lesions, cardiomegaly, trauma, or diaphragmatic hernia was seen. Thoracic ultrasound was performed using GE Logiq 200 Pro Series ultrasound with a 6.5 mHz frequency transducer. A small amount of residual anechoic pleural fluid was present and no pericardial fluid was seen. No mass lesions were seen. At this time, no fibrin or compartmentalization of fluid was evident. Echocardiography was also performed using GE Logiq 200 Pro Series ultrasound with a 7.0 mHz transducer with the cat in right lateral recumbency over a cut out in an examination table. B-mode and M-mode long and short axis cardiac images were obtained (Figure 2). Cardiac measurements were taken on M-mode images at the level of the chordai tendinea on short axis images. Care was taken to ensure perpendicular placement of the cursor to the left ventricular free wall and septum. All views were normal, with no evidence of cardiomyopathy, valvular disease, thrombi, or heartworm infestation (Table 1). The abdomen was scanned with a 6.5 mHz ultrasound probe; no fluid was seen and all abdominal structures were within normal limits. The diaphragm appeared intact, with no evidence of herniation of abdominal viscera. Thoracic fluid analysis (including fluid cholesterol and triglyceride levels), aerobic and anaerobic culture, and cytology were submitted to an outside laboratory for analysis. A complete blood count, serum biochemistry profile including a T4 and free T4 by equilibrium dialysis, feline leukemia virus antigen and feline immunodeficiency antibody testing, and a heartworm antigen and antibody were submitted to an outside laboratory. A urinalysis was performed in hospital. The cat was hospitalized overnight in an oxygen cage as a precaution while diagnostic tests were pending. As the cat was eating and drinking normally and appeared well-hydrated, no further treatment was instituted that day.

FIGURE 2: ECHOCARDIOGRAM: SHORT AXIS B-MODE AND M-MODE IMAGES

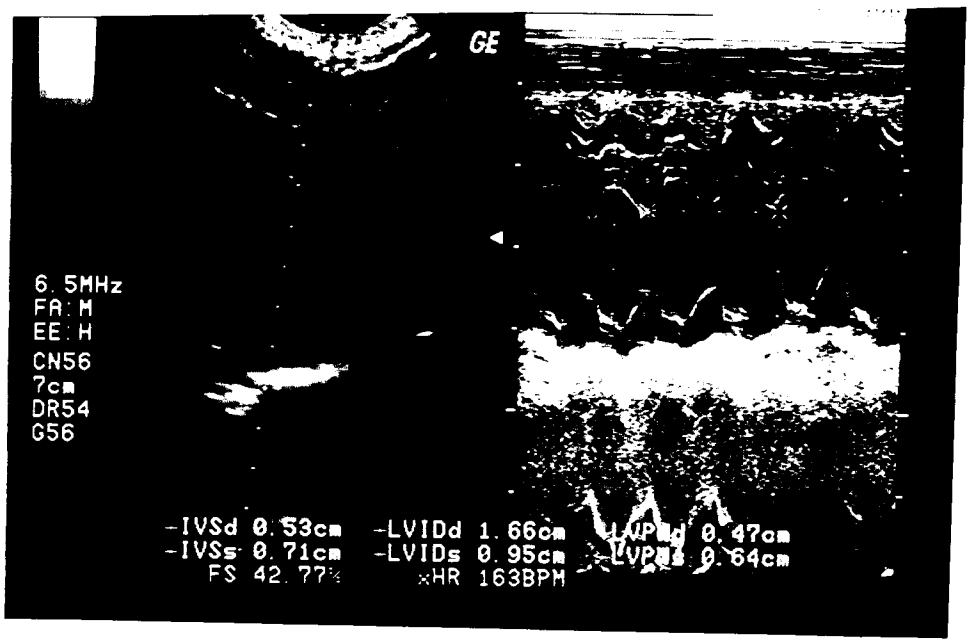

TABLE 1: ECHOCARDIOGRAM

Date	Parameter	Value	Normal Range
1/9/02	IVSTD (interventricular septum at end diastole)	0.53 cm	0.30-0.60 cm
1/9/02	LVIDD (left ventricular internal dimension at end diastole)	1.66 cm	1.08-2.14 cm
1/9/02	LVPWD (left ventricular posterior wall at end diastole)	0.47 cm	0.25-0.60 cm
1/9/02	IVSTS (interventricular septum at end systole)	0.71 cm	0.40-0.90 cm
1/9/02	LVIDS (left ventricular internal dimension and end systole)	0.95 cm	0.40-1.12 cm
1/9/02	LVPWS (left ventricular posterior wall at end systole)	0.64 cm	0.43-0.98 cm
1/9/02	FS (fractional shortening)	42.77%	40.0-67.0%
1/9/02	LA (left atrium)	1.42 cm	0.70-1.70 cm
1/9/02	Ao (aorta)	1.02 cm	0.60-1.21 cm
1/9/02	LA / Ao ratio	1.39	0.88-1.79
1/9/02	Heart rate	171 beats/minute	120-200 beats/minute

The following day, the cat was bright and alert, with normal respiratory pattern, rate (27 breaths per minute) and thoracic auscultation. He continued to be normo-thermic (temperature 38.2°C) and was eating well. Fluid analysis was consistent with chylothorax – the fluid cholesterol:triglyceride ratio was 0.07 (normal >1), the fluid triglyceride to serum triglyceride ratio was 8.97 (normal <1), total protein was 3.8, the nucleated cell count was 5,600 / ul, and fluid culture failed to yield growth (Table 2). The cytological diagnosis was lymphcyte-rich modified transudate with lipid-laden macrophages. No neoplastic cells were noted. The complete blood count values were normal except for moderate lymphopenia (Table 3). Serum biochemistries including T4 and fT4 by ED were within normal limits (Table 3). Heartworm antigen and antibody tests were negative, as were serum feline leukemia antigen and feline immunodeficiency antibody tests. Urinalysis showed normal urine concentration (USG 1.051), with the remainder of the urinalysis and microscopic examination within normal limits (Table 4). Based on these results and the earlier imaging studies, a diagnosis of idiopathic chylothorax was made. The cat was discharged with a low fat diet (Hill's Science Diet r/d canned and dry) and rutin 250 mg every eight hours. The owner was instructed to watch the cat closely for dyspnea and to return for a recheck examination and radiographs in five days.

Two days after discharge, the cat was presented for increased respiratory effort. On physical examination, the respiratory rate was 60 breaths per minute with an increased abdominal effort. Heart sounds and lung sounds were muffled ventrally. The cat's temperature continued to be normal (38.1°C), and the rest of the examination was unremarkable. Thoracic ultrasound was repeated with the cat in ventral recumbency to take advantage of the acoustic window provided by the fluid. Again, thoracic structures appeared normal except for a large volume of pleural fluid. Thoracocentesis was repeated, and 200 ml of chyle were removed. The cat's respiratory rate decreased to 16 breaths per minute following thoracocentesis. The cat was hospitalized for the day for observation, and was released later that day. Referral for surgical thoracic duct ligation was discussed with the owner and was declined. Prednisolone, 2.5 mg every 24 hours, was instituted in an attempt to minimize fibrosing pleuritis.

TABLE 2: FLUID ANALYSIS

Date	Test	Laboratory Value	Normal Reference
1/10/02	Color	Pink-white / opaque	NA
1/10/02	Specific gravity	1.034	NA
1/10/02	WBC count	5,600 / ul	NA
1/10/02	RBC count	4,000 / ul	NA
1/10/02	Protein	3.8 mg/dl	NA
1/10/02	Fluid cholesterol	140 mg/dl	75-220 mg/dl
1/10/02	Fluid triglyceride	1973 mg/dl	25-160 mg/dl
1/10/02	Fluid cholesterol:triglyceride ratio	0.07	>1
1/10/02	Serum triglyceride	220 mg/dl	25-160 mg/dl
1/10/02	Fluid triglyceride: serum triglyceride ratio	8.97	<1

TABLE 3: BLOOD TEST RESULTS

Date	Test	Laboratory Value	Normal Reference
1/10/02	ALT	28 IU/L	10-100 IU/L
1/10/02	ALP	21 IU/L	6-102 IU/L
1/10/02	T. Bilirubin	0.1 mg/dl	0.1-0.4 mg/dl
1/10/02	Total protein	7.7 g/dl	5.2-8.8 g/dl
1/10/02	Albumin	3.6 g/dl	2.5-3.9 g/dl
1/10/02	Globulin	4.1 g/dl	2.3-5.3 g/dl
1/10/02	A/G ratio	0.9	0.35-1.5
1/10/02	Cholesterol	176 mg/dl	75-220 mg/dl
1/10/02	BUN	33 mg/dl	14-36 mg/dl
1/10/02	Creatinine	2.3 mg/dl	0.6-2.4 mg/dl
1/10/02	BUN/Creatinine ratio	14	4-33
1/10/02	Phosphorus	5.6 mg/dl	2.4-8.2 mg/dl
1/10/02	Calcium	10.8 mg/dl	8.2-10.8 mg/dl
1/10/02	Glucose	133 mg/dl	64-170 mg/dl
1/10/02	Sodium	158 mEq/L	145-158 mEq/L
1/10/02	Potassium	4.1 mEq/L	3.4-5.6 mEq/L
1/10/02	Na/K ratio	39	32-41
1/10/02	Chloride	121 mEq/L	104-128 mEq/L
1/10/02	CPK	154 IU/L	56-529 IU/L
1/10/02	WBC count	9.2 x 1000/UL	3.5-16.0 x 1000/UL
1/10/02	RBC	9.8 x 100,000/UL	5.92-9.93 x 100,000/UL
1/10/02	HGB	15.3 g/dl	9.3-15.9 g/dl
1/10/02	PCV	46%	29-48%
1/10/02	MCV	43 fl	37-61 fl
1/10/02	MCH	14.5 pg	11-21 pg

1/10/02	MCHC	34%	30-38%
1/10/02	Absolute neutrophils	7820/UL	2500-8500/UL
1/10/02	Absolute lymphocytes	920/UL	1200-8000/UL
1/10/02	Absolute eosinophils	460/UL	0-1000/UL
1/10/02	Absolute monocytes	258/UL	0-600/UL
1/10/02	Absolute basophils	0/UL	0-150/UL
1/10/02	Quantitative platelet	217 x 1000/UL	200-500 x 1000/UL
1/10/02	T3 (RIA)	48 ng/dl	40-150 ng/dl
1/10/02	T4 (RIA)	1.34 ug/dl	0.8-4.0 ug/dl
1/10/02	FT4 by ED	35.2 pmol/L	10-50 pmol/L
1/10/02	Heartworm antibody	Negative	Negative
1/10/02	Heartworm antigen	Negative	Negative
1/10/02	FeLV Antigen	Negative	Negative
1/10/02	FIV Antibody	Negative	Negative

TABLE 4: URINALYSIS RESULTS

Date	**Test**	**Laboratory Value**	**Normal Reference**
1/10/02	Color	Yellow, clear	Yellow, clear
1/10/02	Urine pH	6.21	5.5-7.5
1/10/02	Urine specific gravity	1.051	>1.040
1/10/02	Urine protein	Negative	Negative
1/10/02	Urine blood	Negative	Negative
1/10/02	Urine ketones	Negative	Negative
1/10/02	Urine bilirubin	Negative	Negative
1/10/02	Urine glucose	Negative	Negative
1/10/02	Urine sediment	None	None

The cat presented similarly every two to three days for the next twelve days. Each time thoracocentesis was performed alternating left and right sides, with 100 to 175 ml of chyle recovered. Surgical treatment was again discussed with the owner and declined. On day eight, aerobic and anaerobic culture of thoracic fluid was repeated to rule out infection due to repeated thoracocentesis. The cultures were negative. The cat continued to eat well, but lost 0.25 kg during the twelve days following presentation. The diet was changed to Hill's Science Diet w/d to decrease calorie restriction while still providing a restricted-fat diet. A decision to place a chest tube was made after thoracocentesis was performed five times. CBC and serum chemistry were performed in-hospital prior to the procedure and were within normal limits except for moderate lymphopenia (Table 5). An IV catheter was placed in the left cephalic vein, and Normosol-R was administered at a rate of 10 ml/kg/hr. The cat was pre-medicated with 1.5 mg butorphanol given subcutaneously. Anesthesia was induced with sevofluorane administered by mask and the cat was intubated. Blood pressure, ECG, and pulse oximetry were monitored throughout the procedure and remained normal (systolic blood pressure > 110 mm Hg, pulse rate 140 to 170 beats per minute, and oxygen saturation greater than 90%). The left lateral thoracic wall was clipped and surgically prepared over the third to ninth intercostal spaces. A local block of the skin, subcutaneous tissue, and intercostal muscles over the sixth and seventh intercostal spaces was performed with 0.2 cc of 2% lidocaine diluted to a total volume of 0.5 cc at the site of planned chest tube placement. A cap, mask, and sterile gloves were worn, and sterile drapes were placed. A small skin incision was made over the mid-thorax at the seventh intercostal space. A 16 french red rubber feeding tube was prepared with the distal aspect attached to a three-way stopcock, extension tubing, and a 60 cc syringe, with all connections wired together to prevent accidental dislodgment. The proximal end of the tube was grasped in a large curved hemostat and burrowed cranially through the subcutaneous tissues to the sixth intercostal space, where the end was forced through the intercostal muscles. The tube was advanced in a cranial-ventral direction into the thorax and secured with a Chinese finger-trap suture. Triple antibiotic ointment was placed at the skin incision and a light non-restrictive bandage was placed. One hundered seventy five milliliters of chyle were aspirated. Radiographs confirmed proper placement of the chest tube in the cranial-ventral thorax and showed nearly complete drainage of fluid, with normal expansion of the lungs. The cat recovered normally

TABLE 5: BLOOD TEST RESULTS – IN HOUSE, PRE-ANESTHETIC

Date	Test	Laboratory Value	Normal Reference
1/22/02	ALT	28 IU/L	10-100 IU/L
1/22/02	ALP	21 IU/L	6-102 IU/L
1/22/02	T. Bilirubin	0.1 mg/dl	0.1-0.4 mg/dl
1/22/02	Total protein	7.7 g/dl	5.2-8.8 g/dl
1/22/02	BUN	33 mg/dl	14-36 mg/dl
1/22/02	Creatinine	2.3 mg/dl	0.6-2.4 mg/dl
1/22/02	Phosphorus	5.6 mg/dl	2.4-8.2 mg/dl
1/22/02	Calcium	10.8 mg/dl	8.2-10.8 mg/dl
1/22/02	Glucose	133 mg/dl	64-170 mg/dl
1/22/02	Sodium	159 mEq/L	145-158 mEq/L
1/22/02	Potassium	4.1 mEq/L	3.4-5.6 mEq/L
1/22/02	WBC count	9.2 x 1000/UL	3.5-16.0 x 1000/UL
1/22/02	RBC	9.8 x 100,000/UL	5.92-9.93 x 100,000/UL
1/22/02	HGB	15.3 g/dl	9.3-15.9 g/dl
1/22/02	PCV	46%	29-48%
1/22/02	MCV	43 fl	37-61 fl
1/22/02	MCH	14.5 pg	11-21 pg
1/22/02	MCHC	34%	30-38%
1/22/02	Absolute neutrophils	7820/UL	2500-8500/UL
1/22/02	Absolute lymphocytes	920/UL	1200-8000/UL
1/22/02	Absolute eosinophils	460/UL	0-1000/UL
1/22/02	Absolute monocytes	258/UL	0-600/UL
1/22/02	Absolute basophils	0/UL	0-150/UL
1/22/02	Quantitative platelet	217 x 1000/UL	200-500 x 1000/UL

from anesthesia, and 0.05 mg of buprenorphine was administered subcutaneously on recovery and repeated every twelve hours while the tube remained in place.

The cat was maintained in the hospital for the next eight days. Intravenous fluids (Normosol-R supplemented with 20 mEq KCl/L) were administered at maintenance rates (66 ml/kg/day) for the first twenty-four hours after chest tube placement, then discontinued as the cat continued to eat and drink normally. Fluid aspiration from the tube was performed daily (Table 6). For the first four days, 80 to 120 ml of fluid were recovered daily. The amount then decreased each day, until on days seven and eight, less than 30 ml of fluid were removed. Thoracic radiographs were repeated on day seven and showed minimal fluid in the thoracic cavity, with good lung expansion. The cat was weighed daily prior to thoracic fluid aspiration, temperature was measured twice daily, and respiratory and pulse rates were measured three times daily (Table 6). The number of calories consumed daily was also calculated (Table 6). Maintenance caloric requirements were calculated to be 316 Kcal/day utilizing the following equation: 1.4 x{(30 x weight in kg)+70}. Buprenorphine, 0.05 mg, was administered subcutaneouly every twelve hours for pain control. The dosage of prednisolone was decreased to 2.5 mg every other day. Rutin, 250 mg, was administered every eight hours. Prophylactic antibiotics were not administered, but daily in-hospital fluid examination was performed looking for the presence of bacteria or neutrophils that could signal infection.

As indicated in Table 6, weight loss continued despite consumption of adequate daily calories, probably due to leakage and removal of large volumes of chyle. Hydration and water consumption was adequate. Therefore, the decision was made on day four (1/26/02) to change from Hill's w/d to a higher calorie diet (Eukanuba kitten dry, a/d canned) to try to further increase caloric intake and decrease weight loss. A low-fat diet is commonly recommended for the treatment of chylothorax but has not been proven to be beneficial in decreasing chyle production. This diet change was successful in promoting increased caloric intake and weight gain without increasing chyle production in this cat. On day eight (1/30/02), thoracic radiographs were repeated and showed minimal pleural effusion and good expansion of all lung lobes. The chest tube was removed and the tip was submitted for aerobic and anaerobic culture. The culture was negative. The cat was discharged from the hospital, with recheck examinations scheduled every five

TABLE 6. MEASURED PARAMETERS DURING HOSPITALIZATION

Date	Volume Aspirated Fluid (ml)	Average Respiratory Rate (bpm)	Average Heart Rate (bpm)	Temp. (°C)	Weight (kg)	Calories consumed (kcal/d)
1/23/02	120 ml	35	170	38.1	5.15	320
1/24/02	88 ml	40	165	38.0	5.10	325
1/25/02	110 ml	40	160	38.1	5.00	325
1/26/02	92 ml	35	160	38.2	4.90	315
1/27/02	75 ml	30	170	38.2	4.85	375
1/28/02	60 ml	25	170	38.1	4.95	380
1/29/02	28 ml	25	165	38.2	5.10	380
1/30/02	10 ml	25	160	38.0	5.41	380

days. The owners were instructed to monitor for dyspnea. Prednisolone, 2.5 mg every other day, was continued to attempt to prevent fibrosing pleuritis. Rutin, 250 mg every eight hours, was also continued.

Over the next month, the cat continued to require thoracocentesis every five to seven days. Sixty to one hundred milliliters of chyle were aspirated each time. One month after removal of the chest tube it was noted that dyspnea and tachypnea (respiratory rate 60 breaths per minute) persisted after bilateral thoracocentesis. Thoracic radiographs showed rounding of the lung borders with minimal residual pleural fluid. Fibrosing pleuritis was suspected. At this time, the author became aware of a study evaluating the efficacy of octreotide, a synthetic somatostatin, in the treatment of idiopathic chylothorax at University of Wisconsin Madison School of Veterinary Medicine via a search of the Veterinary Information Network (VIN). The study protocol recommended octreotide, 10 mcg/kg subcutaneously every eight hours for a minimum of two weeks[a]. The protocol was discussed with the owners of the cat, and informed consent to try this experimental treatment was obtained. The cat was hospitalized for the initial twenty-four hours of treatment to observe for any adverse reaction. Fifty-five mcg octreotide was administered subcutaneously every eight hours. No adverse reactions were observed, and the cat was released to the owners the following day with directions to continue the octreotide injections at home and return for follow-up in five days. Prednisolone, 2.5 mg every other day, and rutin, 250 mg every eight hours, were also continued. The owners were instructed to observe the cat for at least 45 minutes after each injection for increased dyspnea, lethargy, vomiting, or any other problems that may indicate an adverse reaction.

In five days, the cat was presented for a recheck examination. The owners reported no problems noted at home and the cat continued to eat well. The cat had gained 0.2 kg, and weighed 5.61 kg. The cat was normothermic (temperature 38.1 °C), with mild dyspnea (respiratory rate 60 breaths per minute) with a slight abdominal lift on inspiration. Lung sounds were normal. Thoracic radiographs showed a moderate amount of pleural fluid with rounded lung borders. Bilateral thoracocentesis was performed, and 80 ml of chyle was removed. The respiratory effort decreased to almost normal, and the respiratory rate decreased to 45 breaths per minute. A CBC and chemistry panel were submitted to an outside laboratory and were

within normal limits except for moderate lymphopenia (Table 7). The cat was discharged with instructions to continue octreotide, prednisolone, and rutin as previously prescribed and to return in one week.

The following week, the cat presented for follow-up. He weighed 5.64 kg, and was afebrile (temperature 38.0°C), and had mild dyspnea (respiratory rate 46 breaths per minute with a mild abdominal lift on inspiration). The remainder of the physical examination was normal. Thoracic radiographs showed minimal pleural effusion and rounded lung borders. Due to the minimal amount of fluid and mild dyspnea, thoracocentesis was not performed. It was recommended to continue the octreotide, prednisolone, and rutin as prescribed and return for a recheck in two weeks. The owners were also instructed to monitor the respiratory rate at home and return if it increased above 60 breaths per minute at rest.

It was three weeks before the cat was presented for examination. The owners had administered octreotide and prednisolone as directed; however, they had discontinued the rutin due to difficulty of administration. The owners felt the cat was doing very well at home and exhibited minimal breathing difficulty. Physical examination was unremarkable except for mild dyspnea (respiratory rate 40 breaths per minute with mild abdominal lift). Thoracic radiographs showed minimal pleural fluid with rounding of the lung borders. The owners declined bloodwork at this time. After consultation with the chief researcher on the octreotide study at University of Wisconsin Madison, it was decided to discontinue octreotide[a]. The prednisolone was also discontinued, as it appeared that there was now minimal chyle leakage into the pleural space. Monthly rechecks were recommended.

The cat was presented every six to eight weeks for recheck examinations for the next seven months. The owners reported that the cat was doing well at home. His weight remained stable, and his physical examination was unremarkable except for a subtle abdominal lift on inspiration and a mildly elevated respiratory rate (40 to 50 breaths per minute). Because the cat was doing well clinically, thoracic radiographs and bloodwork were not repeated. Seven months after the completion of octreotide treatment, the cat presented with dyspnea (respiratory rate 100 breaths per minute). Thoracocentesis yielded 150 ml

TABLE 7: BLOOD TEST RESULTS-ONE WEEK AFTER INITIATION OF OCTREOTIDE

Date	Test	Laboratory Value	Normal Reference
3/1/02	ALT	38 IU/L	10-100 IU/L
3/1/02	ALP	18 IU/L	6-102 IU/L
3/1/02	T. Bilirubin	0.1 mg/dl	0.1-0.4 mg/dl
3/1/02	Total protein	7.9 g/dl	5.2-8.8 g/dl
3/1/02	Albumin	3.7 g/dl	2.5-3.9 g/dl
3/1/02	Globulin	4.2 g/dl	2.3-5.3 g/dl
3/1/02	A/G ratio	0.88	0.35-1.5
3/1/02	Cholesterol	90 mg/dl	75-220 mg/dl
3/1/02	BUN	29 mg/dl	14-36 mg/dl
3/1/02	Creatinine	2.1 mg/dl	0.6-2.4 mg/dl
3/1/02	Phosphorus	4.3 mg/dl	2.4-8.2 mg/dl
3/1/02	Calcium	9.7 mg/dl	8.2-10.8 mg/dl
3/1/02	Glucose	165 mg/dl	64-170 mg/dl
3/1/02	Sodium	150 mEq/L	145-158 mEq/L
3/1/02	Potassium	4.1 mEq/L	3.4-5.6 mEq/L
3/1/02	Chloride	115 mEq/L	104-128 mEq/L
3/1/02	CPK	210 IU/L	56-529 IU/L
3/1/02	WBC count	9.9 x 1000/UL	3.5-16.0 x 1000/UL
3/1/02	RBC	8.9 x 100,000/UL	5.92-9.93 x 100,000/UL
3/1/02	HGB	15.1 g/dl	9.3-15.9 g/dl
3/1/02	PCV	43%	29-48%
3/1/02	MCV	43 fl	37-61 fl
3/1/02	MCH	14.5 pg	11-21 pg
3/1/02	MCHC	33%	30-38%
3/1/02	Absolute neutrophils	8300/UL	2500-8500/UL

3/1/02	Absolute lymphocytes	716/UL	1200-8000/UL
3/1/02	Absolute eosinophils	420/UL	0-1000/UL
3/1/02	Absolute monocytes	425/UL	0-600/UL
3/1/02	Absolute basophils	0/UL	0-150/UL
3/1/02	Quantitative platelet	316 x 1000/UL	200-500 x 1000/UL
3/1/02	T3 (RIA)	44 ng/dl	40-150 ng/dl
3/1/02	T4 (RIA)	0.94 ug/dl	0.8-4.0 ug/dl

milky white fluid consistent in appearance with chylous effusion. The owner declined further treatment and the cat was euthanized. A necropsy was declined by the owner.

DISCUSSION

This case of an eight-year-old domestic longhaired cat represents a characteristic presentation of idiopathic chylothorax. This cat was initially successfully managed with medical treatment, although he did succumb to the disease ten months after initial diagnosis.

Pleural effusion was suspected based on the presence of dyspnea and muffled heart sounds on initial physical examination. In retrospect, thoracocentesis should have been performed before thoracic radiographs were taken. Although the cat appeared stable and had a respiratory rate of 46 breaths per minutes, a large amount of fluid was present that could have caused acute decompensation. Although the appearance of the fluid obtained on initial thoracocentesis was suggestive of chylous effusion, fluid analysis, including a fluid cholesterol:triglyceride ratio and fluid to serum triglyceride ratio, was necessary to confirm the diagnosis. Underlying causes of chylothorax were ruled out by negative heartworm antigen and antibody levels, negative tests for feline leukemia and feline immunodeficiency viruses, normal serum T4 and fT4 levels, normal abdominal and thoracic, and echocardiography. Traumatic chylothorax could not be ruled out but was deemed unlikely due to the lack of concurrent injuries and persistence of the effusion beyond a few weeks.

This cat was managed initially with a low fat diet, rutin, and thoracocentesis as needed. Six weeks of this treatment alone did not control the chylous effusion, and fibrinous pleuritis was suspected at this time based on continued dyspnea despite adequate thoracic drainage and rounding of lung borders on radiographs. Prednisolone, 2.5 mg once daily, was instituted twelve days after presentation and was not successful in preventing fibrosing pleuritis. A higher dosage may have been more efficacious, although a higher dosage may also have increased the risk of infection or other side effects. The benefit of prednisolone in the prevention of fibrosing pleuritis has not been studied. Surgical management was discussed early in the course of the disease with the owners but was declined. Early surgical intervention may have prevented the

development of fibrinous pleuritis in this cat. However, surgical correction has a variable and disappointing success rate (20 to 60 percent), and carries significant intra- and post-operative risks.

Traditionally, a low-fat diet is recommended in cases of idiopathic chylothorax. However, as demonstrated in this case, weight loss continued despite consumption of adequate daily calories on a low-fat diet. This weight loss was presumed to be due to nutrient loss via the chylous effusion. Weight loss resolved when a higher caloric density diet was offered. It may not be possible for some cats to consume adequate calories when fed a low-fat diet to offset nutrient loss through chyle leakage. No studies to date have supported the efficacy of a low-fat diet in decreasing chyle production. Therefore, in some cats feeding a diet with increased caloric density and fat content may be indicated to prevent cachexia.

Although the initiation of octreotide treatment and the resolution of chylous leakage corresponded in this cat, it is unknown whether octreotide contributed to the resolution of chylothorax in this case. Idiopathic chylothorax can spontaneously resolve in weeks to months. Octreotide therapy was continued for one month. This duration of treatment was somewhat arbitrarily chosen, as the ideal course of treatment is unknown. This treatment is expensive (in this case the cost was approximately $400 per week), which may limit the duration of therapy. In humans, octreotide therapy is discontinued after resolution of chylothorax. Long-term consequences of octreotide usage, if any, are unknown.

The cause for the recurrence of chylothorax after seven months remission was unknown. It is also unknown if a second course of octreotide would have been beneficial at the time of recurrence. The overall prognosis for idiopathic chylothorax is guarded and it is not known which, if any, medical or surgical treatments result in the longest survival.

SUMMARY

An eight-year-old domestic longhaired cat was diagnosed with idiopathic chylothorax. The cat presented for mild dyspnea. The diagnosis was based on radiographs, fluid analysis, and diagnostics to rule-out underlying causes of chylothorax. The initial management consisted of periodic thoracocentesis, rutin,

low-fat diet, and prednisolone. A chest tube was placed after frequent thoracocentesis failed to adequately control dyspnea. The cat was changed to a higher fat diet when weight loss continued despite caloric intake at maintenance levels. Fibrinous pleuritis was suspected six weeks after initial diagnosis based on continued dyspnea despite adequate thoracic drainage and rounding of lung borders on radiographs. Octreotride was added to the therapy six weeks after initial diagnosis, and chylous effusion resolved one week later. It is unknown what role, if any, octreotide played in this outcome. The cat remained free of chylous effusion for seven months, at which time the effusion returned for unknown reasons.

CHYLOTHORAX REFERENCES

[1] Fossum TW, Forrester SD, Swenson CL, et al. Chylothorax in cats: 37 cases (1969-1989). *J Am Vet Med Assoc* 1991; 198:672-678.

[2] Fossum TW. Chylothorax. *In* August JR, ed. *Consultations in Feline Internal Medicine 4.* Philadelphia: WB Saunders Co, 2001:267-276.

[3] Haider M, Schad H, Mendler N. Thoracic duct lymph and PEEP studies in anesthetized dogs. Effect of a thoracic duct fistula on the development of a hyponcotic-hydrostatic pulmonary oedema. *Intensive Care Med* 1987; 13:278-283.

[4] Cohen RG, DeMeester TR, Lafontaine E. The pleura. *In* Sabiston DC, Spencer FC eds. *Surgery of the chest* 6th ed. Philadelphia: WB Saunders Co, 1995:535-538.

[5] Hawkins EC, Fossum TW. Medical and Surgical Management of Pleural Effusion. *In* Bonagura JD, ed. *Kirk's Current Veterinary Therapy XIII, Small Animal Practice.* Philadelphia: WB Saunders Co, 2000:819-825.

[6] Sherding RG. Diseases of the Pleural Cavity. *In* Sherding RG, ed. *The Cat, Diseases and Clinical Management.* New York: Churchill Livingstone, 1994:1053-1091.

[7] Smeak DD, Kerpsack SJ. Management of Feline Chylothorax. *In* Bonagura JD, ed. *Kirk's Current Veterinary Therapy XII, Small Animal Practice.* Philadelphia: WB Saunders Co, 1995:921-927.

[8]Fossum TW. Chylothorax in cats: Is there a role for surgery? *J Feline Med Surg* 2001; 3(2):73-79.

[9]Fossum TW, Evering WN, Miller MW, et al. Severe bilateral fibrosing pleuritis associated with chronic chylothorax in 5 cats and 2 dogs. *J Am Vet Med Assoc* 1992; 201:317-324.

[10]Suess RP, Flanders JA. Constrictive pleuritis in cats with chylothorax: 10 cases. *J Am Anim Hosp Assoc.* 1994; 30(1):70-77.

[11]Glennon JC, Flanders JA, Rothwell JT, et al. Constrictive pleuritis with chylothorax in a cat: a case report. *J Am Vet Med Assoc.* 1987; 23:539-542.

[12] Fossum TW, Jacobs RM, Birchard SJ. Evaluation of cholesterol and triglyceride concentrations in differentiating chylous and nonchylous pleural effusions in dogs and cats. *J Am Vet Med Assoc* 1986; 188:49-51.

[13]Thompson MS, Cohn LA, Jordan RC. Use of rutin for medical management of idiopathic chylothorax in four cats. *J Am Vet Med Assoc* 1999; 215(3):346-348.

[14]Collard JM, Laterre PF, Boemer F, Reynaert M, Ponlot R. Conservative treatment of postsurgical lymphatic leaks with somatostatin-14. *Chest* 2000; 117:902-905.

[15]Demos NJ, Kozel J, Scerbo JE. Somatostatin in the treatment of chylothorax. *Chest* 2001; 119:964-966.

[16]Markham KM, Glover JL, Welsh RJ, Lucas RJ, Bendick PJ. Octreotide in the treatment of thoracic duct injuries. *Am Surg* 2000; 66(12):1165-1167.

[17]Harpster NK. Chylothorax. . *In* Bonagura JD, ed. *Kirk's Current Veterinary Therapy IX, Small Animal Practice.* Philadelphia: WB Saunders Co, 1986:295-303.

[18]Kerpsack SJ, McLoughlin MA, Birchard SJ, Smeak DD, Biller DS. Evaluation of mesenteric lymphangiography and thoracic duct ligation in cats with chylothorax: 19 cases (1987-1992). *J Am Vet Med Assoc* 1994: 205(5):711-715.

[19]LaFond E, Weirich WE, Salisbury SK. Omentalization of the thorax for treatment of idiopathic chylothorax with constrictive pleuritis in a cat. *J Am Anim Hosp Assoc* 2002;38:74-78.

[20]Jerram RM, Fossum T, Berridge B. The efficacy of mechanical abrasion and talc slurry as methods of pleurodesis in normal dogs. *Vet Surg* 1999; 28:322-332.

NOTATIONS

[a]Personal communication: Dr Gretchan Sicard, University of Wisconsin Madison School of Veterinary Medicine, Madison, Wisconsin