Severe bilateral fibrosing pleuritis associated with chronic chylothorax in five cats and two dogs

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Fibrosing pleuritis is a rarely reported complication of chylothorax in which the pleura becomes diffusely thickened, causing pulmonary atelectasis or restriction of normal lung expansion.\(^1\)-\(^5\) We know of 1 case report of a cat with chylothorax and fibrosing pleuritis.\(^2\) We here report severe, bilateral, fibrosing pleuritis that developed in association with chylothorax in 5 cats and 2 dogs.

The diagnosis of chylothorax was based on measurements of cholesterol and triglyceride values of serum and pleural fluid.\(^6\) All animals in this study had respiratory distress, dyspnea, and muffled heart sounds. The duration of respiratory abnormalities was greater than 6 months in 3 cats and 2 dogs. Slight respiratory distress, which often began as coughing and gagging, had progressed to dyspnea in these animals prior to admission. Echocardiographic analysis of cardiac function and structure was normal in all animals except 1 dog (No. 6) in which pericardial effusion was found, and 1 cat in which a small soft tissue mass was noticed at the right atrioventricular junction (No. 2).

Results of routine hematologic and biochemical screening were normal with the exception of mild nonregenerative anemia in 1 cat (No. 4) and 1 dog (No. 6); mild hypoproteinemia, mild hyaloxy binemia, and moderately high serum alanine transaminase in 1 cat (No. 5); and mild neutrophilia, lymphopenia, hypoproteinemia, hyaloxy binemia, hypocalcemia, and thrombocytopenia in 1 dog (case 6). Results of an ELISA for feline leukemia virus antigen were negative in 3 cats and positive in 2 cats (1 of which had mediastinal lymphosarcoma and has been previously reported\(^7\)). Three of 5 cats were tested for heartworm antigen and were negative; 2 of 5 cats were tested and found negative for feline immunodeficiency virus. Microbial culture results of pleural fluid retrieved by needle thoracentesis, or at surgery, were negative in all animals with the exception of 1 dog (No. 6) in which Serratia spp was found.

Thoracic radiography revealed moderate to severe pleural effusion and atelectasis of several or all lung lobes. In most of the animals, the lungs failed to reexpand after thoracentesis and fluid removal.

A 5-year-old neutered male Himalayan cat (No. 1) was admitted to the veterinary medical center for respiratory distress and intermittent vomiting. Slight respiratory distress had progressed to dyspnea over 18 months despite intermittent treatment with antibiotics, and chylothorax had been diagnosed 2 weeks earlier.

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thickened and roughened by whitish-tan material that was confluent with the pleura. Adhesions were found between the lung lobes and the parietal pleura in the left hemithorax. The left lung lobes were markedly collapsed and appeared as small, smooth, rounded hilar masses (Fig 3). Cut surfaces of the lung lobes were wet and dark red. Diffuse moderate fibrous thickening of the pleura was seen microscopically. Mild to moderate pulmonary edema was found throughout the lungs, and a moderate number of foamy macrophages were within alveolar spaces adjacent to more severely affected areas. Additionally, mild bronchitis and bronchiolitis were characterized by intraluminal neutrophils and macrophages admixed with cell debris.

A 5-year-old neutered male domestic shorthair cat (No. 2) was referred for evaluation of dyspnea and lethargy of 3 weeks' duration. A small (1 × 2 cm) soft tissue mass was noticed on echocardiography at the right atrioventricular junction, suggestive of a right atrial tumor. The cat was euthanatized.

Approximately 140 ml of pinkish-white, opaque fluid was found in the thoracic cavity at necropsy. The cranial lung lobes were reduced to round smooth masses of 1.5 to 2 cm in diameter. The remaining lung lobes were dark red and approximately one-half their normal size. A moderate number of fibrin tags were in the left hemithorax attached to the parietal pleura, and the mediastinum was thickened (5 to 6 cm) and contained a moderate amount of milky fluid. Both bronchi were filled with unclotted blood, and the cut surfaces of the lungs exuded abundant amounts of bloody fluid. Pieces of the diaphragmatic lobes, however, still floated in fixative. Histologically, the cranial lobes were markedly atelectatic with marked thickening of the arterial walls. Multiple lymphoid aggregates, which were focally extensive in areas, were found within the subpleural regions of the lung lobes. Mild, diffuse fibrosis (Fig 4) was apparent throughout these lobes with mild, fibrous thickening of the pleura. In the other lung lobes, chronic fibrosing pleuritis was the primary change and depicted by a moderate to marked fibrous thickening of the pleura in which there were moderate numbers of lymphocytes, macrophages, and neutrophils. Multiple, focal, and mild accumulations of lymphocytes were at the border of the lung parenchyma and the thickened pleura. A locally extensive area of atelectasis with marked thickening of arterial vessel walls was evident. Few lymphocytes, macrophages, and neutrophils surrounded these vessels.

A 3-year-old sexually intact male Shetland Sheepdog (No. 3) was referred for evaluation of severe respiratory distress of 4 days' duration. Chylothorax and fibrosing pleuritis was diagnosed and surgical exploration of the thorax performed. Severe fibrosing pleuritis was present and minimal
normal lung tissue was identifiable. Because of poor prognosis, the dog was euthanatized.

The left lung lobes were markedly collapsed and appeared as rounded hilar masses at necropsy (Fig 5); the right lung lobes were approximately one-half their normal size. The surface of the lung lobes appeared roughened and the pleura appeared diffusely thickened. Histologically, the predominant changes in the lungs were marked diffuse fibrous thickening of the pleura characterized by markedly increased amounts of fibrous connective tissue and vascular proliferation (Fig 6). A mild amount of lymphocytes, histiocytes, plasma cells, and few neutrophils were scattered throughout the connective tissue. Slightly increased amounts of inflammatory cells were found at the junction of the lung parenchyma and pleura. Mesothelial cell hyperplasia was evident in some segments of the pleura, and multiple finger-like projections of fibrous tags with mild inflammatory infiltrates were attached to the pleural surface. A few areas of mild fibrinous exudate, admixed with areas of hemorrhage, were attached to the pleural surface. Mild loosening of perivascular tissue within the lung parenchyma was evident, consistent with edema. Locally extensive areas of mild alveolar collapse were apparent, especially in subpleural areas. A few thrombosed blood vessels were within the lung parenchyma.

A 4-year-old, spayed domestic shorthair cat (No. 4) was referred for evaluation of lymphadenopathy, lethargy, anorexia, and dyspnea of 2 weeks' duration. The cat was FeLV positive and biopsy of the left cranial cervical lymph node confirmed the diagnosis of lymphoblastic lymphosarcoma. Despite radiographic disappearance of the mediastinal mass following chemotherapy, the chylothorax continued and frequent thoracenteses were required to prevent dyspnea. Perforated plastic sheeting6 was placed bilaterally in the diaphragm as previously described.8 The caudal lung lobes visualized through the diaphragmatic incisions appeared normal. During the next 4 months the owners reported that the cat occasionally became dyspneic; however, the dyspnea was relieved when the owner held the cat in an upright position. The cat was euthanatized 4.5 months after surgery because of complications associated with recurrence of lymphosarcoma.

6Medical grade Silastic sheeting, Dow Corning Corp, Midland, Mich.
At necropsy, generalized lymphadenopathy was present and fibrous adhesions were noted between the diaphragmatic surface of the liver and the plastic mesh. These adhesions appeared to severely limit the patency of the plastic mesh. A 3 x 6 cm homogenous mediastinal mass overlay the tracheal bifurcation. On sectioned surface, the mass was determined to be a lymph node. The visceral pleura of all lung lobes was diffusely whitish-tan, moderately thickened, and roughened. Both caudal lung lobes were adhered to the diaphragm and the parietal pleura. All lung lobes had dark-red cut surfaces and were contracted to various degrees.

Histologic examination confirmed the diagnosis of disseminated lymphoblastic lymphosarcoma involving multiple lymph nodes, liver, and small intestine. In the lungs, the predominant change was one of a chronic fibrosing pleuritis, characterized by diffuse moderate to marked thickening of the pleura by fibrous connective tissue, with moderate infiltrates of lymphocytes, macrophages, and plasma cells. The inflammation was present in varying intensity throughout the thickened pleura. A few fibrin tags were on the surface of the pleura. A thin layer of lymphocytes and few macrophages were at the junction of the pleura and lung parenchyma. Mild to moderate perivascular edema was apparent throughout the lungs.

A 3-year-old spayed domestic shorthair cat (No. 5) was referred for evaluation of chylothorax of 8 months' duration and acute dyspnea and lethargy of 3 weeks' duration. Despite needle thoracentesis and removal of 275 ml of chylous fluid, the left cranial, right cranial, and right middle lung lobes remained collapsed (Fig 7).

Surgery was performed and perforated plastic sheeting placed bilaterally in the diaphragm. The caudal lung lobes, visualized through the diaphragmatic incisions, appeared atelectatic and compressed and the pleura appeared thickened. The cat remained severely dyspneic and died despite intensive postoperative care 72 hours after surgery. A necropsy was not performed.

A 2-year-old sexually intact female mixed-breed dog (No. 6) was referred for evaluation of chylothorax, subcutaneous chyle, limb and neck edema, dyspnea and chylothorax of 6 months' duration. Microbial culture results were negative. Despite dietary management, intermittent thoracentesis, and intermittent antibiotic therapy, the dog became increasingly dyspneic and forelimb and neck edema developed, from which a milky white fluid exuded. At admission, the dog was extremely dyspneic and cachectic. Muffled lung and heart sounds were heard bilaterally, and the jugular veins appeared distended. All limbs were edematous, most markedly in the front limbs. Subcutaneous swelling was also noticed ventrally, extending from the thoracic inlet to the 13th ribs. Fluid oozed from the skin overlying the left ventral portion of the thorax.

Lymphoscintigraphy revealed an abnormally high rate of diffusion of radiolabel throughout the tissues. Cytologic evaluation of the thoracic fluid revealed degenerative neutrophils and numerous intracytoplasmic rod-shaped organisms. Microbial culture yielded *Serratia* spp. Antibiotic therapy was initiated on the basis of sensitivity testing; however, the dog died 2 days after admission.

Necropsy revealed approximately 500 ml of turbid, light brown fluid within the thoracic cavity. The parietal pleura was diffusely roughened and brown with numerous, moderately strong, tan to red fibrous strands attaching it at random to the lungs, pericardial sac, and mediastinum. The lungs were shrunken to about one-fourth their normal size and were diffusely dark red and moderately firm. Their cut surfaces were diffusely red and slightly wet. The pericardium contained approximately 150 ml of whitish-tan fluid. Multiple, light gray, small (1 to 2 mm), friable, granular structures were evident within the thoracic and pericardial exudates. Cytologic examination of these structures revealed bacterial colonies.

Microscopically, the predominant change was that of severe chronic fibrous pleuritis characterized by a marked amount of fibrin attached to fibrous connective pleural tissue and admixed with hemorrhage, proteinaceous exudate, and moderate numbers of neutrophils, macrophages, and lymphocytes. Fibroblast proliferation was moderate throughout the pleura. Multiple gram-negative bacterial colonies were trapped within the exudate.
Chylothorax is a debilitating, often fatal, disease of cats and dogs that has been recognized with increasing frequency in the last few years. The cause of the effusion is frequently not apparent; however, it has been diagnosed in association with cardiomyopathy, cranial mediastinal masses, heartworm disease, trauma, intestinal lymphangiectasia, congenital abnormalities of the thoracic duct, and generalized lymphangiectasia with subcutaneous chyle leakage. Clinical signs often develop in animals for months or years prior to diagnosis of chylothorax. Both medical and surgical treatment of chylothorax have been recommended. Medical management (tube thoracentesis and a low fat diet), which is intended to decrease chyle formation while the thoracic duct heals, has lost favor in recent years with the finding that trauma is a relatively rare cause of chylothorax. However, the most widely accepted surgical treatment of this disease, thoracic duct ligation, is often unsuccessful, even when combined with mesenteric lymphangiography. Consequently, a number of other procedures have been introduced in recent years, which are designed to palliate the clinical signs associated with massive pleural effusion by directing the effusion into the venous system (pleurovenous shunting) or the abdominal cavity (pleuropertitoneal shunting). The latter may be either active, using commercial shunting devices (Denver double-valve peritoneo-venous shunt or Hakim-Cordis pediatric ventricular-peritoneal shunt), or passive, via instillation of diaphragmatic mesh. These procedures are not designed to terminate the production of chyle, they merely palliate the clinical signs associated with pleural effusion. The effect of chronic effusions on the pleura have seldom been addressed in the veterinary literature.

The animals in this report all had severe diffuse bilateral fibrosing pleuritis, which developed in association with chronic chylothorax. The terms restrictive and constrictive have previously been used to identify this condition. We have elected to use the term fibrosing pleuritis because both constriction of the pulmonary parenchyma and restriction of pulmonary expansion occurred in these animals.

Bilateral fibrosing pleuritis is an infrequently reported condition in human beings associated most commonly with tuberculosis, empyema, and asbestos exposure. It has also been associated with rheumatoid effusions, uremia, pancreatitis, traumatic hemotherax, and chylothorax. In rare cases, no underlying cause can be found. Several drugs have been implicated as causing pleural fibrosis in human beings, including propranolol and methysergide. Pyothorax, chylothorax, feline infectious peritonitis, hemotherax, and tuberculosis have been associated with the development of this condition in animals. Six animals in this study had chylothorax only; 1 dog also had pyothorax, which may have contributed to the pleural changes. Although the cause of the fibrosis is unknown, it apparently can develop subsequent to any prolonged exudative or blood-stained effusion. Exudates are characterized by a high rate of fibrin formation and degradation.

and Serratia spp were cultured from this fluid. The lung parenchyma was diffusely congested, and multiple, subpleural areas of minimal hemorrhage and fibrinous exudate were apparent.

A 5-year-old neutered male domestic shorthair cat (No. 7) was referred for evaluation of dyspnea and chylothorax. Abnormally rapid and shallow respirations were first noticed 9 months earlier. Chylothorax had been diagnosed 2 weeks prior to referral.

A median sternotomy was performed, and a thick solid sheet of fibrous tissue was found directly under the sternum. The lungs and heart could not be identified. Blunt dissection was used to remove the fibrous tissue until the pulmonary parenchyma and pericardium could be identified. Pleural fluid was minimal, but the lung lobes were markedly collapsed. The right cranial lung lobe was 2 cm in diameter. A 2.5-mm-thick fibrous tissue covered the visceral surfaces of all lung lobes, parietal pleura, and pericardium. A combination of sharp and blunt dissection was used to gently remove the fibrous tissue from the underlying pulmonary parenchyma. The fibrous tissue was not firmly adherent to the lung tissue and in most places could be removed with a sterile swab. In a few areas damage to the underlying lung tissue resulted in pneumothorax and mild hemorrhage. Following decortication, the edges of the lung lobes appeared rounded but otherwise normal, and pulmonary expansion was markedly improved. The left caudal lobe was adhered to the diaphragm and an automatic stapling device was used to perform a partial lobectomy. A chest tube was placed and the sternotomy closed. Approximately 1 hour following completion of surgery harsh lung sounds were heard. Pulmonary edema was suspected and diuretic therapy instituted. Despite aggressive therapy, the cat died 4 hours after surgery. A necropsy was not performed.

Samples of fibrous tissue overlying the parietal and visceral pleura were submitted for histologic examination. The tissue overlying the visceral pleura was composed of granulation tissue and mature fibrous connective tissue. Fibrinous trabeculae lined by fibroblasts were evident in the superficial areas. Hypertrophic mesothelial cells lined the tissue in a segmental distribution; some areas were devoid of mesothelial cells. A mild number of neutrophils, macrophages, lymphocytes, and plasma cells were spread diffusely, especially in areas of granulation tissue; tissue overlying the parietal pleura was similar, except that there were a moderate number of vessels.

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Inflammatory exudates, such as chylothorax and pyothorax, induce changes in mesothelial cell morphologic features, resulting in increased permeability, mesothelial cell desquamation, and triggering of both pathways of the coagulation cascade. These desquamated mesothelial cells have also been shown to produce type-III collagen in cell culture, promoting fibrosis. Additionally, chronic presence of pleural fluid might lead to an impairment in the mechanism of fibrin degradation. Fibrinolysis may decrease because direct injury to mesothelial cells may reduce inherent fibrinolytic activity of the cells, and/or the increased fluid volume may dilute local plasminogen activator. Plasminogen activator converts the precursor plasminogen to its active form, plasmin. Fibrinolytic activity in mammals is attributable primarily to this serine protease.

In animals with fibrosis, the pleura is thickened by diffuse fibrous tissue that restricts normal pulmonary expansion. Pulmonary function testing in human patients with fibrosing pleuritis has shown a decrease in vital capacity and static compliance, necessitating greater negative intrapleural pressures for any given change in lung volume, compared with healthy patients. In 1 cat with chylothorax, pulmonary dysfunction associated with severe atelectasis, interstitial fibrosis, and accumulation of fibrin within the alveoli contributed to persistent respiratory distress despite apparent expansion of the lungs following decortication; the cat was euthanatized 5 days after surgery. In the animals of this report, the degree of fibrosis was such that ventilatory abnormalities (as signified by an inability of the lungs to expand normally), appeared to either contribute to the animals' natural death, or signified such a poor prognosis that euthanasia was recommended.

The volume of pleural effusion in these animals was minimal and alone would not have accounted for the severity of the dyspnea that was evident clinically. The combination of fibrosing pleuritis and pulmonary edema may have contributed to the dyspnea. The only effective treatment for fibrosing pleuritis is decortication. Decortication gives the best functional result when the pleuritis is of short duration and pulmonary parenchymal disease is minimal. Therefore, it is recommended that decortication be performed as soon as possible after the diagnosis. Apparently, as the pleural peel matures, blood vessels may extend into the underlying pulmonary parenchyma, making decortication difficult. Decortication in human beings carries a good prognosis if only 1 or 2 lobes are involved; however, when the fibrosis is diffuse, as in the animals of this report, even with effective decortication a guarded prognosis is warranted. Harpster reported that lung expansion and pulmonary function will improve over a 2- to 3-month period after decortication. Corticosteroids may be beneficial initially and for 2 to 4 weeks after decortication. We have found in animals with less severe fibrosing pleuritis than reported here, and in 1 of the cats of this report, that the thickened pleura is not firmly adherent to the underlying parenchyma and can be removed without severely damaging the underlying lung; however, pneumothorax is a common sequelae after decortication and usually requires tube thoracentesis. Mechanical pneumothorax is encouraged in those animals in which the parietal and visceral pleura may be beneficial, and chronic chylothorax may also have been associated with undiagnosed chronic chylothorax. Potentially, animals with chylothorax remain relatively free of clinical signs as long as pleural fluid resorption equals or exceeds fluid production. Lack of clinical signs of dyspnea may delay the diagnosis in these animals. Two additional animals had confirmed chronic chylothorax; they became progressively more dyspneic with time. Restrictive pulmonary disease probably contributed to the increased dyspnea. The cases reported here represent 70% of the cases of dogs and cats with chylothorax referred to the authors' institution during this study period; however, because of selection bias, this figure does not accurately estimate the prevalence of fibrosing pleuritis in animals with chylothorax.

One animal in this study developed restrictive pleuritis after placement of plastic sheeting in the diaphragm for passive pleuroperitoneal drainage. The long-term result of pleuroperitoneal or pleurovenous shunting procedures on the development of this condition is unknown, but warrants further research. Fibrosing pleuritis may be a serious and life-threatening long-term complication associated with drainage procedures if chylothorax.

The atelectasis in 5 animals was so severe that some lung lobes (particularly the cranial lobes) appeared as rounded hilar masses; these masses were confused with neoplasia radiographically and on echocardiography in 1 cat. Similar masses have previously been reported in a cat with chylothorax. Round, pleural-based masses, usually found in the posterior surface of a lower lung lobe, have been reported in human beings with fibrous pleural thickening. The condition is referred to as rounded atelectasis, or pseudotumor. The pathogenesis of rounded atelectasis remains obscure, but it has been suggested that the atelectatic lung develops in association with pleural effusion, and the fibrous pleuritis develops secondarily. Others...
believe that the atelectasis results from contraction of collagen that occurs during the development and maturation of the fibrous peel. In some animals of this report, the pleura was thicker in the lung lobes that were only partially collapsed and still ventilating, than in those that were totally collapsed, making the former hypothesis more plausible. Histologically, the underlying lung may or may not have interstitial fibrosis. Mild interstitial fibrosis was found in the collapsed lobes of 2 cats and a dog in this report and in 1 cat in another report. It is not known whether fibrosis developed secondarily to the atelectasis, or contributed to its formation. However, the degree of fibrosis in some totally collapsed lobes was such that pulmonary reinfation may not have occurred after decortication.

The cat in which decortication was performed appeared to develop pulmonary edema after surgery and died. Pulmonary edema has been reported as a complication of reexpansion of atelectatic lung lobes and is potentially a serious complication of decortication and pulmonary reexpansion in animals with fibrosing pleuritis. Three other animals in this study had mild to moderate pulmonary edema. Histologic evidence of perivascular edema in some animals was believed to be related to the pathologic process, because fluid in animals euthanatized by use of barbiturates usually seeps into alveoli, as opposed to the interstitium. The pathogenesis of the diffuse interstitial fibrosis apparent in 1 case is also not known, but longstanding edema has been known to result in fibrosis of alveolar walls in human beings. It is therefore possible that mild and chronic interstitial edema could have a role in the disease process. Careful monitoring of animals with fibrosing pleuritis for evidence of pulmonary edema is warranted and care should be taken to avoid rapid reexpansion of atelectic lobes.

Diagnosis of fibrosing pleuritis is difficult. The atelectatic lobes may be confused with metastatic or primary pulmonary neoplasia, lung lobe torsion, or hilar lymphadenopathy. Radiographic evidence of pulmonary parenchyma that fails to reexpand after removal of pleural fluid should be considered possible evidence of atelectasis with associated fibrosis. Fibrosing pleuritis should also be considered in animals with persistent dyspnea, yet minimal pleural fluid. In human beings, computerized tomography is occasionally helpful in differentiating pleural fibrosis from other abnormalities, such as neoplasia. In most of the animals of this report, the amount of pleural effusion was overestimated radiographically. The thickened pleura and atelectatic lobes were believed to represent moderate to severe pleural effusion; however, in some of these animals, only minimal pleural fluid was evident at surgery or necropsy.

Diffuse, fibrosing pleuritis may severely decrease the animals ability to ventilate, warranting an extremely guarded prognosis. Studies in dogs and cats have determined that coughing may be the only clinical sign of chylothorax. Because early recognition of this disease is essential, animals with nonspecific respiratory tract signs should have thoracic radiography done to determine whether effusion is present. The propensity for the development of fibrosing pleuritis should be considered when chylothorax is initially diagnosed and early attempts to terminate the production of chyle appear to be warranted.

22. Suter PF, Greene RW. Chylothorax in a dog with ab-

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