Buffy the Chylothorax Cat

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Introduction

Chylothorax is an uncommon disease affecting dogs and cats characterized by accumulation of chyle within the thoracic cavity. The earliest papers discussing chylothorax in dogs and cats were written in the 1950s, though successful treatment strategies still remain elusive (6). Studies have implemented thoracic duct ligation and pericardectomy and report success rates reaching 80%-100%, though many other studies have had poor success rates (1,7). Animals should be fully evaluated for an underlying disease, but many times the chylothorax is diagnosed as idiopathic (6). The pathophysiology of idiopathic chylothorax is poorly understood. No superior medical protocol has emerged, but it is initially recommended since the condition may resolve spontaneously and surgical options require a high level of expertise (1,6).

Medical management is available and the therapies aim to either remove chyle from the thorax or reduce the production of chyle. Reduction of chyle production is attempted through the utilization of low-fat diets, Rutin, furosemide, antibiotics, and steroids (6). The goal of surgical therapy is to stop the accumulation of chyle in the thoracic cavity. The primary procedure performed to achieve this goal is thoracic duct ligation (TDL) completed via thoracotomy. Adjunct procedures performed with TDL include omentalization, decortication of constrictive pleuritis, pericardectomy, cisterna chyli ablation and active pleuroperitoneal shunts (1, 3, 4).

History and Presentation

Buffy, an 11-year-old spayed DSH, presented to MSU-CVM on 01/04/2017 for previously diagnosed chylothorax. Buffy began losing weight the beginning of in early December 2016 and exhibited labored breathing starting around December 12, 2016. Buffy's owner was concerned and brought her to the rDVM on 12/13/2016, who performed thoracic radiographs and a thoracocentesis. A second thoracocentesis was performed on 12/15/2016 and

the fluid was sent off for analysis. Buffy was diagnosed with chylothorax and prescribed Rutin, 250 mg twice daily, on 12/16/2016. A third thoracocentesis was performed on 12/19/2016, and the fourth thoracocentesis was performed on 12/28/2016. After the fourth thoracocentesis, Buffy's owner reported that she did not respond as well as she had before and was open-mouth breathing. Buffy had been doing okay at home, though she had continued to open-mouth breathe after excitement along with the continued weight loss.

Upon presentation, Buffy was bright, alert, and responsive. She had a temperature of 103.1 F, pulse of 200, and a respiratory rate of 80. She weighed 3.9 kg with a body condition score of 4 out of 9. She had an abnormal and shallow breathing pattern but the rest of her physical exam was within normal limits.

Diagnostic Approach

A small animal profile was completed and all values were within normal limits. The CBC revealed a mild anemia (PCV 27%; RI 30-46), and a mild lymphopenia (440 /uL ;RI: 1200-6500). Ultrasound-guided thoracocentesis was performed and 90 mL of an opaque beige fluid was obtained. This fluid was sent for analysis and contained 4,410/uL nucleated cells, 4.6 g/dL protein, 50% non-degenerate neutrophils, 45% small lymphocytes, and was determined to be an exudate with mixed inflammation consistent with a chylothorax. The effusion from the chest was tested for triglyceride levels and compared to the triglyceride levels of the serum. The fluid triglyceride level was 1115 mg/dL and the serum triglyceride level was 39 mg/dL, consistent with chylothorax.

Thoracic radiographs were taken, which revealed severe pleural effusion and pneumothorax. There was a mediastinal shift to the left, likely secondary to atelectasis. There was a mass effect likely artifact due to the pleural effusion. There was convex margination and

soft tissue swelling of the left lateral thoracic wall musculature due to edema or cellulitis following thoracocentesis or granulomatous disease. An echocardiogram was performed, demonstrating a moderate amount of pleural effusion consistent with previously noted chylothorax, no evidence of heart disease, normal appearance of the pericardium.

A CT of the thorax with contrast revealed a moderate amount of pleural effusion within the ventral and left aspect of the thorax consistent with neoplastic effusion, exudate, chyle, or modified transudate. There was a large amount of gas within the pleural space of the right hemithorax and small amount within the left hemithorax due to either thoracocentesis or necrosis of the lung. There was a structured interstitial pulmonary pattern characterized by multiple sharply marginated, soft tissue dense strongly contrast enhancing masses in all lung lobes. Differentials for these masses included metastatic neoplasia, granulomas, or abscesses. There was no normal lung tissue within the right and left cranial lung lobes. The volume of the left caudal lung lobe was severely decreased. There was a soft tissue swelling and thickening of the intercostal musculature of the left lateral body wall at the 5^{th}-7^{th} intercostal spaces due to cellulitis or edema following thoracocentesis or neoplasia. There was a mediastinal shift to the left likely secondary to the decreased lung volume and atelectasis. Pectus excavatum was also present.

On 01/05/2017, an ultrasound-guided fine-needle aspirate was obtained from the mass seen on radiograph and CT. Findings included small amounts of a thick basophilic proteinaceous fluid and moderate numbers of normal appearing small lymphocytes revealing thoracic fluid contamination or lymphocytic inflammation, consistent with lymphocytic fluid.

Pathophysiology

The most common diagnosis of chylothorax is idiopathic, though other causes include trauma, heartworm disease, cardiomyopathy, neoplasia, cranial mediastinal masses, venous thrombosis, fungal granulomas, congenital thoracic duct abnormalities, and constrictive pericarditis (2, 6). Chylothorax has occurred in several species including bovine, canine, and feline, however, this disease process is relatively uncommon in cats (2).

Food is digested and dietary fat components are broken down into small molecules called chylomicrons. Chylomicrons predominantly consist of triglycerides, phospholipids, and cholesterol (5). These are collected by villous lacteals and emptied into the cisterna chyli, which is the abdominal lymphatic reservoir within the craniodorsal abdomen. This intestinal lymph called “chyle” is distinctly opaque and milky-white in appearance though can be clear, pinkish, or yellowish in color (3). Chyle is comprised of proteins, dietary fats, fat-soluble vitamins, electrolytes, and cells of the immune system. The cranial extension of the cisterna chyli is the thoracic duct, which is the largest lymphatic vessel in the body.

The three roles of the lymphatic system are to maintain fluid balance, generate an immune response, and perform uptake and transport of the dietary fats (5). Pathological processes or abnormality in the thoracic duct can prevent the flow of chyle into the cranial vena cava, increasing lymphatic pressure, and cause chyle to leak into the thoracic cavity from the vessels (2). These pets have difficulty breathing as the chyle builds up in the chest preventing their lungs from fully inflating with air. Chyle is an irritant and chronic presence within the thoracic cavity can lead to inflammation of the heart and lungs causing fibrosing pleuritis and pericarditis (1, 5).

Treatment and Management

On 01/06/2017 thoracoscopy was performed, using a paraxyphoid camera portal in dorsal recumbency and a 5 mm scope. Thoracic exploratory was performed, but was hindered by severe pleural adhesions. A celiotomy was performed along with a transdiaphragmatic thoracotomy. Methylene blue dye (0.1 ml) was injected in a mesenteric lymph node to help visualize the thoracic duct. The thoracic duct identified dorsal to the aorta in the caudal thorax and ligated with hemostatic clips. The cisterna chyli was then ablated. A chest tube was placed intraoperatively to allow for post-operative fluid removal within the thoracic cavity and an esophagostomy tube was placed to provide Buffy with nutrition during post-operative recovery. She was started on buprenorphine 0.02 mg/kg IV Q8, Cerenia 1 mg/kg IV Q24, cefazolin 22 mg/kg IV Q8, Terbutaline 0.01 mg/kg SQ Q8, and Albuterol 180 mcg PRN.

Buffy did not recover well from anesthesia. Before leaving radiology at 2 pm her pCO2 reading was 90 mm Hg. After arrival in ICU her anesthetic drugs were reversed but she seemed to still be heavily sedated. She was placed in the oxygen cage and was closely monitored. Her blood gas was measured again at 5 pm and included a pCO2 of 96 mm Hg. She was bolused with 30 mL LRS and 10 mL of hypertonic saline. She was also nebulized with 180 mcg of Albuterol. Her blood gas was measured again and the pCO2 was at 68 mm Hg. Her temperature had dropped to 95 F and she was provided supplemental heat. Buffy was then placed back into the oxygen cage. Her pCO2 reading at 10 pm was back to 85.8 mm Hg and she was again nebulized with 180 mcg of Albuterol. She was closely monitored overnight for dyspnea and other signs of discomfort. Fluid accumulation quantified by removal from the thoracic drain averaged 12.1 ml/kg/day for the first day. Buffy remained in the oxygen cage with constant ECG monitoring.

On 01/07/2017 the fluid aspirated from her chest doubled in volume to 23.5 ml/kg/day. She was breathing well on her own and she was started on esophagostomy tube feeding at 25% RER. Small animal profile was completed, with abnormalities including ALT 83 U/L (7-60), ALP 8 U/L (10-42), TBili 0.6 mg/dL (0.1-0.5), total protein 4.2 g/dL (6.5-8.4), Albumin 1.5 g/dL (2.2-3.2), Globulin 2.7 g/dL (4.1-6.0), Calcium 7.6 mg/dL (8.2-10.6). She continued to receive intravenous fluid LRS at 13 mL/hr and received buprenorphine 0.02 mg/kg IV Q8, Cerenia 1 mg/kg IV Q24, and Terbutaline 0.01 mg/kg SQ Q8. Buffy remained in the oxygen cage with constant ECG monitoring with regular SpO2 and blood pressure checks.

The evening of 01/07/2017, PCV was checked due to concerns of pale gums and was 42%, with lipemic serum. We suspected she was beginning to develop hepatic lipidosis due to this along with the slight change in liver values. She was bolused IV fluids of 15 mL/kg and fluids were increased to 16 mL/hr. Pleural fluid aspirated was serosanginous and red tinged and her fluid production was 38.5 mL/kg/day. She was still breathing well on her own and increased her enteral feeding to 50% RER. Buffy received Buprenorphine 0.02 mg/kg IV Q8, Cerenia 1 mg/kg IV Q24, and Terbutaline 0.01 mg/kg SQ Q8. Buffy remained in the oxygen cage with constant ECG monitoring with regular SpO2 and blood pressure checks.

On 01/09/2017, a CBC was completed revealing a PCV of 25% (RI 30-46) and continued lymphopenia (158 /uL;RI: 1200-6500). The thoracic fluid aspirated from her chest tube was suddenly dark red in color and had a PCV 3% and TP 1.4 g/dL. A coagulation panel was submitted to evaluate her clotting factors and this revealed prolonged PT of 17.1 seconds (5-10 sec) and decreased PTT of 13.3 seconds (15-25 sec). She continued to rest comfortably in her cage and was maintained at 50% RER for her esophagostomy tube feeding. Later that day, Buffy

went into sudden cardiac arrest. After consulting with the owner, Buffy was euthanized, and her body submitted for necropsy.

Necropsy Findings

On necropsy, the thoracic cavity contained 100 mL of reddish white opaque fluid. The lungs were markedly atelectic and most of the lung lobes were reduced to small round balls covered by thick white tissue. The same thick white tissue covered the pericardium and widened the mediastinum. The left cranial and caudal lung lobes were completely bridged by thick layers of white fibrous tissue. The liver was mildly enlarged and the intestinal tract was within normal limits although largely empty.

The thoracic wall was examined and the parietal pleura was irregularly thickened by a wide layer of fibrovascular tissue consisting mostly of fibrosis with slender fibrocytes and fibrillary collagen interspersed with small numbers of blood vessels. The pancreas had multiple foci of necrosis interstitially and effacing pancreatic lobules evident of acute pancreatitis. This was accompanied by large numbers of neutrophils, many degenerate. The lung covered by dense white tissue had very thick layers of collagen rich and cell poor connective tissue along the peripheral margin. The lung parenchyma was totally compressed and alveolar septal walls could not be distinguished.

The right caudal and accessory lung lobes had pleural fibrosis but aerated well and had increased numbers of alveolar macrophages seen in alveoli. The right cranial, middle, and caudal lung lobes had foci of profound atelectasis in which there was transition between markedly condensed and atelectic areas and areas with mild interstitial edema or interstitial septal thickening. The left cranial lung lobe had profound atelectasis, entrapping thick pleural fibrosis with intermingled lymphocytes and fibrosis with bronchial gland hyperplasia.

The moderate to severe fibrosing pleuritis present was due to long-standing chylous effusion which affected visceral and parietal pleura. The right cranial and middle, left cranial, and most of left caudal lung lobes were more completely compressed and nonfunctional, mostly due to atelectasis with some consideration for interstitial fibrosis. There were some increased peribronchial lymphocytes seen. The lung lobes were rounded and had obvious thick white tissue covering the visceral pleura. The lobes most severely affected were incapable of re-expansion. The lymphocyte nature of chyle is likely associated with inflammatory cytokines that drive the pleural fibrosis.

Conclusion

Buffy had a chronic history of weight loss and restricted breathing which were only temporarily alleviated using thoracocentesis performed by her rDVM. Diagnostics performed at MSU-CVM including bloodwork, pleural effusion analysis, thoracic radiographs, and CT confirmed Buffy was suffering from chylothorax. These diagnostics revealed atelectasis within multiple lung lobes, pneumothorax, and the negative effects of the serial thoracocenteses she received. Thoracic duct ligation with cisterna chyli ablation was performed but pleural effusion remained to be an issue post-operatively. The chronicity of Buffy's chylothorax led to restrictive pleuritis, which negatively affected her prognosis and outcome. She was also suffering from acute pancreatitis evident at the time of necropsy and this would have been difficult to manage alongside her recovery from her thoracoscopy.

Managing an idiopathic chylothorax patient can be frustrating, as evideced by this case. It can be postulated the chronicity of the chylothorax for this patient was the main reason for the negative outcome. This patient had limited functional lung tissue and was unable to adequately compensate for this deficit post-operatively. The chronicity of the chylothorax led to severe

atelectasis and restrictive pleuritis which were not resolved by performing the thoracic duct ligation. For those cases which chylothorax is diagnosed and treated promptly, prognosis is improved because the negative chronic inflammatory effects of chyle in the thoracic cavity have not yet developed.

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