Idiopathic primary chylopericardium in a dog

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Case Description—A 7-year-old spayed female Labrador Retriever was evaluated because of pericardial effusion.

Clinical Findings—The dog had a history of decreased appetite and exercise intolerance of 3 days’ duration. Thoracic radiography performed by the referring veterinarian revealed a large cardiac silhouette. Heart sounds were muffled. Echocardiographic findings were indicative of severe pericardial effusion with cardiac tamponade; no pleural effusion was identified. Pericardiocentesis yielded a considerable amount of chylous fluid. A diagnosis of chylopericardium in the absence of pleural effusion was made.

Treatment and Outcome—Conservative management was not effective, and subtotal pericardectomy and thoracic duct ligation were recommended. Surgery was postponed by the owners for 25 days, at which time the dog had both chylopericardium and chylothorax. The dog underwent subtotal pericardectomy and thoracic duct ligation; to delineate the thoracic duct, intraoperative lymphangiography was performed by injection of a radiopaque contrast agent directly into a mesenteric lymph node and subsequent injection of methylene blue solution into another mesenteric lymph node. Surgical treatment resulted in complete resolution of the clinical signs and pleural effusion.

Clinical Relevance—To the authors’ knowledge, this is the first report of the development of chylopericardium prior to development of chylothorax in a dog. Treatment with thoracic duct ligation and pericardectomy resulted in complete resolution of the effusion and clinical signs.

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ABBREVIATIONS

OVC-VTH Ontario Veterinary College Veterinary Teaching Hospital

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A 7-year-old spayed female Labrador Retriever was evaluated at the OVC-VTH because of pericardial effusion. The dog had a history of decreased appetite and exercise intolerance of 3 days’ duration. Immediately prior to examination by the referring veterinarian, the dog had an episode of collapse after chasing a cat. Thoracic radiography performed by the referring veterinarian revealed a markedly large cardiac silhouette, consistent with pericardial effusion. On initial evaluation (day 1) at the OVC-VTH, the dog’s rectal temperature and heart rate were within reference limits and it was panting. Cardiac auscultation revealed decreased heart sounds. Mean arterial blood pressure (determined indirectly) was 76 mm Hg. Echocardiography was performed and revealed severe pericardial effusion with cardiac tamponade. There was no evidence of a heart-base mass.

Pericardiocentesis was performed, and 760 mL of chylous fluid was removed from the pericardial sac. Thoracic radiography revealed that the cardiac silhouette was still abnormally large. Pericardiocentesis was repeated, which yielded 360 mL of similar chylous fluid. After this second procedure, a decrease in the size of the cardiac silhouette was apparent radiographically. The pericardial fluid was submitted for cytologic examination, microbial culture, and assessment of protein and triglyceride concentrations. Cytologic findings were consistent with a modified transudate; a mild inflammatory response (primarily macrophages [44%] and lymphocytes [34%]) was evident. The WBC count was 4.0 × 10⁶ cells/L, and the protein concentration was 39 g/L. Microbial culture of the fluid yielded negative results. The triglyceride concentration in the fluid was 3.86 mmol/L. At this initial evaluation, blood samples were collected for a CBC and serum biochemical analyses; assessment of serum revealed that the triglyceride concentration was 0.38 mmol/L. The fluid triglyceride-to-blood triglyceride concentration ratio was 10.1; this, combined with the cytologic findings, was consistent with chylous effusion.

Results of the CBC were within reference limits except for mild lymphopenia (0.78 × 10⁶ cells/L; reference range, 0.8 to 5.1 × 10⁶ cells/L). Results of the serum biochemical analyses were unremarkable except for mildly high sodium concentration (156 mmol/L; reference range, 140 to 134 mmol/L) and mildly high chloride concentration (123 mmol/L; reference range, 104 to 119 mmol/L). Coagulation tests were performed, and results were within reference limits.

Twenty-four hours after the initial evaluation, radiography revealed no signs of further accumulation of fluid in the pericardial or pleural space. A subtotal pericardectomy and thoracic duct ligation were recommended. The owners elected to take the dog home to consider their options. At the time of discharge, the heart and lung sounds were readily ausculted in all fields.

Twelve days after the initial evaluation, the dog was returned to the OVC-VTH because of exercise intolerance and mild dyspnea. Abnormalities detected during physical examination included weak peripheral pulses, muffled heart sounds bilaterally, and a high respiratory rate. Echocardiographic findings confirmed severe pericardial effusion and cardiac tamponade. Pericardiocentesis was performed, and 450 mL of chylous fluid was removed from the pericardial space.
Pericardectomy was again recommended, but the owners elected to continue the conservative treatment. The dog was discharged on the same day, and the owners were instructed to administer prednisone orally at a tapering anti-inflammatory dose (1 mg/kg [0.45 mg/lb], PO, q 24 h for 7 days, then 0.3 mg/kg [0.14 mg/lb], PO, q 24 h for 7 days).

Twenty-five days after the initial evaluation, the dog was returned for examination because of clinical signs similar to those identified previously. On physical examination, the dog was dyspneic with an increased abdominal component to its breathing pattern; respiratory rate was 98 breaths/min. Mucous membranes were pale. Heart sounds were muffled, as were lung sounds; the latter could be ausculted only in the dorsal lung fields, which was suggestive of pleural effusion. Pleural effusion was identified radiographically. A thoracic tube was placed on the left side at the seventh intercostal space, and 1 L of chylous fluid was removed. Echocardiography revealed mild pericardial effusion. There were no signs of cardiac tamponade, and pericardiocentesis was not performed. A CBC and serum biochemical analyses were performed, and findings were unremarkable except for mildly low concentrations of total protein (51 g/L; reference range, 55 to 74 g/L) and globulin (18 g/L; reference range, 21 to 42 g/L). The dog remained in the intensive care unit for 3 days for drainage of the pleural space via the thoracic tube.

Twenty-eight days after the initial evaluation, the dog underwent pericardectomy and thoracic duct ligation. The dog was placed in left lateral recumbency to allow access to the entire right aspect of the thorax and flank. Following aseptic preparation, a flank approach was made to the abdomen caudal to the 13th rib and a mesenteric lymph node was located and exteriorized. Lymphangiography was performed by injecting 6 mL of an iodinated radiopaque contrast agent (diatrizoate meglumine 50% and diatrizoate sodium 25%) directly into the lymph node. Because the ideal interval between injection and radiographic assessment was unknown, lateral radiographic views of the thorax were obtained 90, 150, 210, 270, and 330 seconds after injection of contrast agent into the lymph node. A high-quality lymphangiogram, in which the thoracic duct was outlined, was attained 150 seconds after injection into the lymph node. The flank incision was temporarily closed by use of towel clamps. A right lateral thoracotomy at the fourth intercostal space and subphrenic pericardectomy were performed. The heart was palpated and examined for evidence of a heart-base mass; no abnormalities were detected. The resected portion of pericardium was submitted for histologic evaluation; no abnormalities were detected. A second right lateral thoracotomy was performed at the 10th intercostal space. The flank incision was opened again to allow access to another mesenteric lymph node. On this occasion, 0.8 mL of new methylene blue solution was injected directly into the lymph node; as a result, the thoracic duct at the caudal thoracotomy site was located and ligated with 2 circumferential ligatures of 2-0 monofilament, nonabsorbable suture material and several hemostatic clips. A third mesenteric lymph node was located, and lymphangiography was repeated by use of the aforementioned technique; 130 seconds after injection of contrast agent into the lymph node, a radiographic view of the thorax was obtained. Because no contrast agent was detected cranial to the ligation site, successful ligation of the thoracic duct was confirmed. The 3 surgical approaches were closed routinely.

The dog recovered uneventfully in the intensive care unit. Analgesia was provided via a constant rate infusion of fentanyl (2 to 8 µg/kg/h [0.9 to 3.6 µg/lb/h], application of a fentanyl patch (75 µg/h), and IV administration of meloxicam (0.1 mg/kg [0.45 mg/lb/24 h]). The thoracic tube that was placed before surgery was maintained; a small amount of fluid and air was drained from the thorax. After surgery, the amount of fluid drainage decreased rapidly, and the thoracic tube was removed 24 hours later. The dog was discharged from the hospital 3 days after surgery; at that time, it was considered clinically normal. The dog was returned to the OVC-VTH 5 weeks after surgery. Clinically, the dog appeared normal, with normal appetite, activity level. Thoracic radiography revealed a slight pleural fissure line but no other abnormalities. During the subsequent 2 years of follow-up, the dog remained clinically and radiographically normal.

Discussion
Although there are multiple case reports of primary idiopathic chylopericardium in the human medical literature, this is, to the author’s knowledge, the first report of the development of primary idiopathic chylopericardium (prior to the development of chylothorax) in a dog. In humans, the treatment typically involves open or thoracoscopic ligation of the thoracic duct and partial pericardectomy and usually results in resolution of the disease. There is 1 case report in the human medical literature of primary chylopericardium in an infant that was successfully treated via drainage and medical management only.

The underlying cause of idiopathic chylopericardium in any species is unknown. The etiology is likely similar to that of idiopathic chylothorax, which is not well understood. In clinically normal dogs, the mesenteric lymphatic vessels coalesce at the cisterna chyli. From there, lymph is channeled cranially through the diaphragm, continuing cranially into the thoracic duct. The thoracic duct then drains into the cranial vena cava, and the lymph enters the systemic circulation. In instances of chylothorax, lymph leaks from the lymphatic vessels in the thorax and is free in the pleural space. In idiopathic chylothorax, this is likely a result of abnormalities such as lymphangiectasia in the lymphatic vessels. Presumably, the cause of chylopericardium is similar, with the exceptions that the abnormal lymphatic vessels are contained within the pericardium and that there is a connection between the thoracic duct and the pericardial lymphatic system. It is possible that chylopericardium is a self-perpetuating condition because the pericardial effusion may lead to an increase in right-sided venous pressure, which would prevent the forward flow of chyle into the cranial vena cava.
In humans, it has been suggested that the etiology of primary chylopericardium involves lymphangiectasia, the presence of an anatomic communication between the thoracic duct and the lymphatic vessels of the pericardial sac, and damage to the valves in the thoracic duct.\textsuperscript{3-6} This anatomic connection between the thoracic duct and the pericardial sac has been well supported in the human medical literature in case reports\textsuperscript{7,8} of idiopathic chylopericardium. Imaging modalities that have been used to evaluate humans with primary idiopathic chylopericardium include enhanced computed tomography combined with lymphangiography\textsuperscript{9,10} and lymphscintigraphy\textsuperscript{11,12}. These imaging techniques were not performed in the dog of this report. An anatomic connection between the thoracic duct and pericardial sac, similar to that involved in lymphangiectasia, was not detected in the dog of this report via lymphangiography. However, this is likely attributable to limitations of the lymphangiographic procedure rather than the absence of a connection between these 2 structures.

It is interesting that current recommendations for the treatment of chylothorax in dogs and cats also include thoracic duct ligation and pericardectomy; for which there is a reported success rate of 90%.\textsuperscript{13} It has been hypothesized that an increase in right-sided venous pressure develops as a result of chylothorax-induced pericardial thickening. This increase in pressure contributes to the leakage of chyle into the thorax because it increases the resistance to contents of the thoracic duct entering the right-sided venous system.\textsuperscript{14-16} In the dog of this report, a pericardectomy was indicated both to palliate the pericardial effusion and, possibly, to treat an underlying factor of the disease. The need for thoracic duct ligation was questionable in the dog of this report but was performed in light of the fact that this procedure appears to be the standard management for successful resolution of this disease in humans.\textsuperscript{17,18} Furthermore, there was a case report\textsuperscript{19} of primary chylopericardium in a human for whom treatment involved thoracic duct ligation and pericardectomy; that treatment failed to resolve chyle production. However, a postoperative lymphangiogram revealed a duplicate thoracic duct that was not ligated during the initial surgery. A second surgery to ligate that duct resulted in resolution of the chylous effusion.\textsuperscript{19} Also, treatment failures that occurred in the case series reported by Fossum et al\textsuperscript{10} resolved after thoracic duct ligation. Currently, thoracic duct ligation is the recommended treatment for chylothorax in dogs and cats.

The dog of this report was initially evaluated because of pericardial effusion but developed chylovous pleural effusion during the course of treatment. This may have been a result of repeated pericardiocenteses; those procedures may have created a rent in the pericardium that allowed fluid to accumulate in the thorax instead of the pericardium. If this occurred, some palliation of the cardiac tamponade would be expected. This may explain why the interval between the second and third evaluations was longer than the interval between the first and second evaluations. The development of chylothorax may also have been attributable to a natural progression of the disease that affected lymphatic vessels in the pericardium first and then lymphatic vessels in the thorax. Because chylothorax developed secondary to pericardial effusion and cardiac tamponade in the dog of this report, this finding could also support the theory that an increase in right-sided venous pressure can contribute to the development of chylothorax.

To the authors’ knowledge, this is the first report of clinical use of direct injection of a mesenteric lymph node with contrast material for purposes of lymphangiography; however, the procedure performed in dogs under experimental conditions has been reported by Brisson et al\textsuperscript{19} in that study\textsuperscript{19}, the most useful lymphangiograms were attained 60 to 120 seconds after injection of the lymph node. In the dog of this report, the most useful lymphangiogram was attained 150 seconds after injection of the lymph node. In both the experimental setting\textsuperscript{19} and clinical situation of this report, the method of direct injection of a mesenteric lymph node for purposes of lymphangiography was technically easier to perform than catheterization of a mesenteric lymphatic vessel and provided a good-quality lymphangiogram. Mesenteric lymph node injection with methylene blue solution for delineation of the thoracic duct has been previously reported\textsuperscript{20} in dogs. In the dog of this report, that technique provided excellent delineation of the thoracic duct. Having identified the thoracic duct, it was successfully ligated. The combination of pericardectomy and thoracic duct ligation provided immediate relief of the cardiac tamponade and long-term resolution of the disease and pleural effusion.

References
Selected abstract for JAVMA readers from the American Journal of Veterinary Research

Effects of carprofen on renal function during medetomidine-propofol-isoflurane anesthesia in dogs
Jan H. M. Frendin et al

**Objective**—To investigate effects of carprofen on indices of renal function and results of serum biochemical analyses and effects on cardiovascular variables during medetomidine-propofol-isoflurane anesthesia in dogs.

**Animals**—8 healthy male Beagles.

**Procedures**—A randomized crossover study was conducted with treatments including saline (0.9% NaCl) solution (0.08 mL/kg) and carprofen (4 mg/kg) administered IV. Saline solution or carprofen was administered 30 minutes before induction of anesthesia and immediately before administration of medetomidine (20 µg/kg, IM). Anesthesia was induced with propofol and maintained with inspired isoflurane in oxygen. Blood gas concentrations and ventilation were measured. Cardiovascular variables were continuously monitored via pulse contour cardiac output (CO) measurement. Renal function was assessed via glomerular filtration rate (GFR), renal blood flow (RBF), scintigraphy, serum biochemical analyses, urinalysis, and continuous CO measurements. Hematologic analysis was performed.

**Results**—Values did not differ significantly between the carprofen and saline solution groups. For both treatments, sedation and anesthesia caused changes in results of serum biochemical and hematologic analyses; a transient, significant increase in urine alkaline phosphatase activity; and blood flow diversion to the kidneys. The GFR increased significantly in both groups despite decreased CO, mean arterial pressure, and absolute RBF variables during anesthesia.