Use of a jugular vein autograft for reconstruction of the cranial vena cava in a dog with invasive thymoma and cranial vena cava syndrome

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- Malignant thymomas are characterized by local invasion of surrounding mediastinal tissue, which may include the cranial vena cava.
- Ultrasound-guided fine-needle tissue biopsy and cytologic examination is helpful in definitive diagnosis of tumor type.
- Contrast-enhanced, helical computed tomography is useful for detection of vascular involvement and a tumor thrombus.
- Resection of the cranial vena cava and an autogenous jugular vein graft may be used for restoration of normal venous return to the right atrium and alleviation of cranial vena cava syndrome.

A 12-year-old 21.4-kg (47-lb) spayed female Australian Shepherd was evaluated at the **Veterinary Medical Teaching Hospital (VMTH)**, University of California, for a 6-week history of intermittent edema of the ventral cervical region, lethargy, cough, and reduced exercise tolerance. The referring veterinarian had identified a grade 1/6 left-sided systolic heart murmur. A CBC and serum biochemistry panel revealed WBC count of 24,500 cells/µL (reference range, 6,000 to 17,000 cells/µL) with 22,050 neutrophils/µL (reference range, 3,000 to 11,500 cells/µL), 245 lymphocytes/µL (reference range, 1,000 to 4,800 cells/µL), 1,715 eosinophils/µL (reference range, 100 to 1,250 cells/µL), albumin concentration of 2.3 g/dL (reference range, 2.6 to 4.3 g/dL), and cholesterol concentration of 377 mg/dL (reference range, 112 to 328 mg/dL). Urinalysis revealed specific gravity of 1.017 with 2+ protein (reference range, negative to trace) and no RBCs, unremarkable sediment, and urine protein-to-creatinine ratio of 3.5 (reference limit range, < 1.0). The remainder of the laboratory results were within reference ranges. Thoracic radiography revealed a soft-tissue opacity cranial to the heart. Treatment had included several corticosteroid injections and orally administered antimicrobials (enrofloxacin and amoxicillin-clavulanic acid), which the owner reported had initially resulted in a decrease in cervical swelling with persistence of lethargy and cough. A low-protein diet,[a] enalapril[b] (0.5 mg/kg [0.23 mg/lb], PO, q 12 h), and aspirin (2 mg/kg [0.9 mg/lb], PO, q 24 h) administration were initiated prior to referral.

Physical examination abnormalities on initial examination at the VMTH included mild edema in the ventral mandibular region. Thoracic auscultation revealed a grade 1/6 left-sided systolic heart murmur and normal bronchovesicular lung sounds in all fields. A CBC, serum biochemical analyses, urinalysis, urine bacteriologic culture, urine protein-to-creatinine ratio, coagulation panel, thoracic radiographs, abdominal and thoracic ultrasonography, and echocardiography were performed. Hematologic abnormalities included leukocyte count of 35,040 cells/µL with 30,835 neutrophils/µL with a mild left shift (1,051 band neutrophils/µL; reference range, 0 to 300 cells/µL) and slight toxic change. Serum biochemical abnormalities included slightly high alkaline phosphatase (142 U/L; reference range, 15 to 127 U/L) and γ-glutamyltransferase (10 U/L; reference range; 0 to 6 U/L) activities, high cholesterol concentration (437 mg/dL), and slightly low albumin concentration (2.3 g/dL). Total serum protein concentration was within the reference range. Urinalysis revealed moderate proteinuria, with a urine protein-to-creatinine ratio of 3.71, unremarkable sediment, and urine specific gravity of 1.023. The remainder of the laboratory test results were within reference ranges.

Thoracic radiographs revealed increased opacity and widening of the cranial mediastinum with a discrete soft tissue structure in the cranioventral portion of the thorax on the right lateral projection. A nonspecific pulmonary interstitial pattern was also observed. Ultrasound examination of the cranial portion of the thorax was performed via intercostal and thoracic inlet windows. Two soft tissue masses of similar echogenicity, 3 and 2 cm in diameter, were identified in the cranioventral portion of the thorax. An aspirate of the larger mass was highly cellular and contained high numbers of neutrophils, low numbers of erythrophagic macrophages, and moderate numbers of mast cells. There were low numbers of partially keratinized squamous epithelial cells with nuclear-to-cytoplasmic asynchrony and disproportionately large nuclei for the degree of keratinization. Large numbers of small lym-

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Supported by Small Animal Surgery Service Teaching Allowance, Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California, Davis, CA 95616.

The authors thank John Doval for technical assistance in image preparation.

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phocytes and rare spindloid cells, singly and in small loose clusters, consistent with thymic epithelial cells[1] were observed. Differential diagnoses include thymoma with associated necrotic inflammation, suppurative lymphadenitis, mast cell tumor, and nonexfoliating nonthymic neoplasia with marked inflammation. Serum was submitted for an acetylcholine receptor antibody assay, and acetylcholine receptor antibodies were not detected. The echocardiographic diagnosis was mild mitral regurgitation attributable to myxomatous mitral valve degeneration, with no indications of endocarditis. Systolic blood pressure measured by noninvasive Doppler sphygmomanometry was 170 mm Hg.

The dog was premedicated with morphine (0.5 mg/kg, IM) and glycopyrrolate (0.01 mg/kg [0.005 mg/lb], IM), and anesthesia was induced with propofol (2 mg/kg, IV) and midazolam (0.3 mg/kg [0.14 mg/lb], IV). An endotracheal tube was placed, and anesthesia was maintained with isoflurane and 100% oxygen. Positive-pressure ventilation was provided for the duration of anesthesia. After induction of general anesthesia, noncontrast and iodinated contrast-enhanced (iothalamate sodium,[c] 66.8% [2.2 mg/lb {1.0 mL/lb}, IV]) thoracic helical computed tomography was performed. Findings included a ventral cranial mediastinal mass with a lobular border and slight contrast enhancement. Distention of the cranial vena cava was apparent, with an extensive contrast void within the caval lumen suggestive of tumor infiltration or thrombus formation (**Figure 1**). The plain radiographic and ultrasound examination performed previously had not identified caval invasion or occlusion. After computed tomography examination, an ultrasound-guided renal biopsy was performed with an 18-gauge biopsy needle.[d] Three tissue samples were collected and submitted for histopathologic examination. Moderate widespread membranous glomerulonephritis with tubular ectasia was detected.

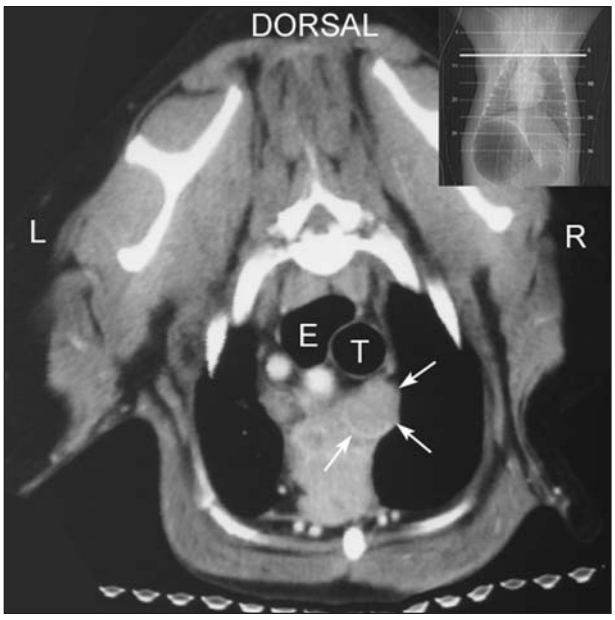

Figure 1—Contrast-enhanced transverse helical computed tomographic image of a ventral thoracic mass with involvement of the cranial vena cava in a dog. Notice extensive lesion indicated by lack of contrast material within the lumen of the cranial vena cava (arrows). T = Trachea. E = Esophagus. L = Left. R = Right.

The dog was prepared for exploratory thoracotomy. Epidural analgesia was performed (preservative-free morphine [0.1 mg/kg {0.05 mg/lb}] diluted in 0.9% NaCl to a volume of 6 mL). Cefazolin (22 mg/kg [10 mg/lb], IV, q 2 h) was administered during surgery. Buccal mucosal bleeding time was determined to be 160 seconds (reference limit, < 300 seconds), and a blood cross-match was performed. Anesthetic monitoring consisted of continuous electrocardiography and measurement of direct and indirect arterial blood pressure, esophageal temperature, end-tidal carbon dioxide concentration, and end-tidal isoflurane concentration. Body temperature was reduced to 32°C (89.6°F). Lactated Ringer's solution was administered at 10 mL/kg (4.5 mL/lb) with 20 mEq of potassium chloride/L. Two units of cross-matched RBCs and 2 units of fresh frozen plasma were administered during the surgical procedure to treat intraoperative hemorrhage and maintain plasma oncotic pressure.

Surgical exploration of the thorax was performed via cranial median sternotomy. A pigmented, lobular 3-cm mass was located in the cranial mediastinum adhered to the right phrenic nerve, right internal thoracic artery, cranial vena cava, and mediastinal tissues. The right internal thoracic artery was ligated with 3-0 silk[e] to allow elevation of the mass. A mildly enlarged, 2-cm diameter lymph node adjacent to the mass was resected. The mediastinal mass was dissected from surrounding structures and noted to be invading the wall of the cranial vena cava. Palpation of the cranial vena cava revealed an 8 × 2-cm intraluminal soft tissue mass. Rumel tourniquets were placed with umbilical tape on the cranial vena cava proximal and distal to the intraluminal mass. After temporary occlusion of the cranial vena cava with the tourniquets, the mediastinal mass and affected portion of the wall of the vessel were resected and an 8 × 2-cm tumor thrombus, which extended caudally through the vessel, was removed. The mediastinal mass and lymph node were submitted for histopathologic examination. The defect in the vessel wall was closed via longitudinal apposition of the resected wall edges with 4-0 waxed[f] silk[g] in a single-layer continuous pattern. Surgical loupes[h] were used for this procedure. When tourniquets were released, the vessel lumen appeared to be attenuated approximately 80% and there was no blood flow apparent through the repaired section. Total occlusion time at this point was 34 minutes.

To reconstruct the surgically attenuated section of the cranial vena cava, an autogenous jugular venograft was harvested. An incision in the midcervical area over the ventral aspect of the left jugular groove was made. The skin and subcutaneous tissue were retracted to expose and isolate the left external jugular vein. Lidocaine was administered topically to the graft vessel to minimize venospasm. An 8-cm portion of jugular vein was harvested for the venograft, and the left external jugular vein was ligated cranially and caudally. The jugular vein diameter was approximately half of the diameter of the nonaffected portion of the cranial vena cava. The 6-cm attenuated portion of the cranial vena

cava was resected en bloc after placement of atraumatic vascular occlusion clamps. The cranial extent of the resected portion was caudal to the confluence of the brachiocephalic veins. The thoracic duct was not seen (it commonly enters the venous system in the vicinity of the junction of the left subclavian and left jugular vein where the brachiocephalic trunk originates). The transected ends of the cranial vena cava were flushed with heparinized 0.9% NaCl solution. For cranial and caudal vascular anastomoses, two 4-0 silk sutures[g] were preplaced and tied at 180° on opposing lateral aspects of the vessel ends of the jugular venograft and cava. The back wall (dorsal aspect) of the vein was sutured in a continuous pattern from an intraluminal approach with 1 of the preplaced sutures. The front wall (ventral aspect) of the vein was then sutured with a continuous pattern, using the other preplaced suture. To compensate for the luminal disparity between the jugular vein and cranial vena cava, the gap between adjacent sutures was greater on the cranial vena cava, compared with the jugular vein. After completion of both suture anastomoses, the occlusion clamps were released and there was good blood flow through the venograft, with minimal, self-limiting hemorrhage apparent at the anastamotic sites. **(Figure 2)**. Total occlusion time for the attempted venotomy repair and subsequent jugular venograft collection and anastomosis was 122 minutes. A 20-F thoracostomy tube[i] was placed, and the incisions in the caudal portion of the neck, sternum, and ventral portion of the thorax were closed routinely.

At the completion of surgery, the dog was actively warmed to 36°C (97.2°F) prior to recovery from anesthesia. Arterial blood gas analysis after extubation revealed mild hypoxemia with Pa_{O_2} of 78 mm Hg and Pa_{CO_2} of 49.6 mm Hg. The calculated Sa_{O_2} was 94%, and the alveolar-arterial gradient was 17 mm Hg. The dog was placed in an oxygen cage at this time with a **fraction of inspired oxygen (F_{IO_2})** of 40%.

Direct arterial blood pressure, rectal temperature, and cardiac rhythm were all monitored continuously. Concentrations of arterial blood gases, serum electrolytes, blood glucose, and serum lactate were measured every 4 hours for the first 12 hours postoperatively. A urethral catheter was placed and connected to a closed urinary collection system.

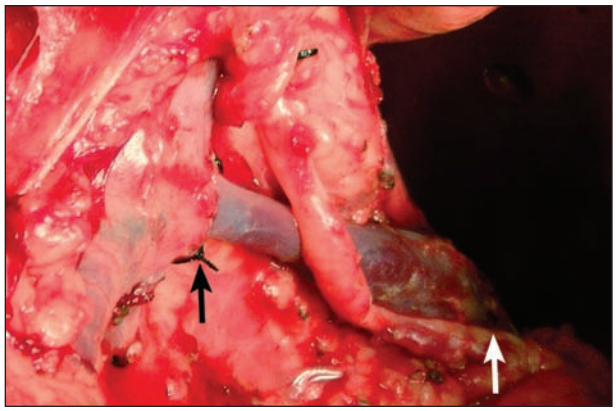

Figure 2—Intraoperative photographic view of a completed venograft in the dog of Figure 1 after removal of a ventral thoracic mass with involvement of the cranial vena cava. Notice cranial anastomosis (black arrow) and caudal anastomosis (white arrow) formed by use of the left external jugular vein, which was sutured to resected ends of the cranial vena cava at the site of tumor resection. The left internal thoracic artery overlies the graft.

Initial postoperative analgesia consisted of morphine (0.5 mg/kg, IV) administration every 4 hours. At 24 hours after surgery, this regimen was replaced with a constant rate infusion of morphine (0.2 to 0.5 mg/kg/h [0.09 to 0.23 mg/lb/h], IV), which was titrated to effect and continued for 3 days. Bupivicaine (0.5% diluted volumetrically 1:3.5 with 0.9% NaCl solution and further diluted 9:1 with sodium bicarbonate solution [8.4%]) was administered intrapleurally via the thoracostomy tube immediately prior to extubation and repeated once 12 hours after surgery. A transdermal fentanyl patch (50 µg/h) was placed 3 days after surgery.

Medical management after surgery consisted of fluid therapy with lactated Ringer's solution (75 mL/h) supplemented with potassium chloride at 30 mEq/L and hetastarch[j] (1 mL/kg/h [0.45 mL/lb/h]). Cefazolin administration was continued (22 mg/kg, IV, q 8 h), and a metoclopramide[k] constant rate infusion (0.02 mg/kg/h [0.009 mg/lb/h], IV) was administered to prevent postoperative ileus. Unfractionated heparin[l] administration (75 U/kg [34 U/lb], SC, q 6 h) was started 24 hours after surgery.

The dog developed interstitial edema of the face, left forelimb, and ventral portion of the thorax within 12 hours after surgery. The edema was treated initially with furosemide administration (1 mg/kg [0.45 mg/lb], IV, q 6 h). The edema resolved 72 hours after surgery, and diuretic administration was discontinued. The thoracic tube was removed 36 hours after surgery when fluid collection rate decreased to < 1 mL/kg per 24 hours. Hematologic abnormalities 36 hours after surgery included marked neutrophilic leukocytosis (39,160 WBCs/µL; reference range, 3,000 to 11,500 WBCs/µL) with a mild left shift (1,760 band neutrophils/µL; reference range, 0 to 300 cells/µL) and no apparent toxic changes. The dog had mild thrombocytopenia (118,000 platelets/µL; reference range, 150,000 to 400,000 platelets/µL), low plasma protein concentration (4.8 g/dL; reference range, 5.4 to 7.5 g/dL), and unremarkable hematocrit (41%; reference range, 40% to 55%). Serum biochemical abnormalities included aspartate aminotransferase activity of 107 U/L (reference range, 15 to 43 U/L), alkaline phosphatase activity of 174 U/L (reference range, 15 to 127 U/L), and albumin concentration of 1.5 g/dL (reference range, 2.9 to 4.2 g/dL).

The dog's blood oxygenation decreased during the 24 hours after surgery (Pa_{O_2}, 70 mm Hg; F_{IO_2}, 60%; and Pa_{CO_2}, 32 mm Hg). The calculated Sa_{O_2} for this Pa_{O_2} was 94.4%, and the corresponding alveolar-arterial gradient was approximately 320. After 24 hours, there was a gradual improvement in oxygenation, and oxygen administration was discontinued on day 5 after surgery.

At this time, the dog was bright, responsive, ambulatory, and eating and drinking voluntarily. A CBC at this time revealed that the WBC concentration had decreased to 16,447 WBCs/µL and the left shift had resolved. The

arterial and urethral catheters were removed, and the dog was transferred to a standard hospital ward.

Six days after surgery, an ultrasound examination of the cranial portion of the thorax revealed normal laminar flow through the venograft and appropriate right atrial filling. Thoracic radiographs and a nonselective angiogram were performed with 20 mL of iopamidol 41%[m] administered via a left cephalic venous catheter. Radiographic findings included mild pleural effusion and pulmonary infiltrates. Iodinated contrast material filled the remaining sections of the cranial vena cava and the jugular venograft, confirming patency of the jugular venograft.

The dog was discharged from the hospital 8 days after surgery. The dog was prescribed enalapril (0.5 mg/kg, PO, q 12 h) to decrease the degree of proteinuria and enoxaparin,[n] a low-molecular-weight heparin (1.0 mg/kg, SC^2, q 6 h, for 1 month), to reduce the risk of venograft thrombosis.

On histologic examination, the mediastinal mass and the mass within the cranial vena cava were composed of dense sheets of neoplastic epithelial cells mixed with variable numbers of well-differentiated lymphocytes and mast cells. The epithelial cells had pale wispy eosinophilic cytoplasm and variably sized nuclei. The histologic diagnosis was a malignant thymoma with intraluminal invasion of the cranial vena cava. On gross and histopathologic examination, the intravascular portion of the mass had a well-organized thrombus closely associated with the submitted tumor tissue. The mediastinal lymph node had marked reduction in the lymphoid cell population consistent with disuse or atrophy caused by compression.

One month after surgery, urinalysis revealed improved urine protein-to-creatinine ratio (1.29) and urine specific gravity of 1.021. The nonselective angiogram was repeated, which revealed patency of the cranial vena cava and jugular venograft. Dosage of enalapril was tapered over the next 2 months and then administration was discontinued. Four months after surgery, the dog appeared clinically normal, was eating and drinking well, and had a normal activity level. Serum biochemical analyses, CBC, and urinalysis were repeated and revealed persistent hypoalbuminemia (2.1 g/dL) and hypercholesterolemia (463 mg/dL). The urine protein-to-creatinine ratio, total protein concentration, BUN concentration, and creatinine concentration were within reference ranges. Thoracic radiographs revealed that cardiovascular and pulmonary structures were within normal limits. Nonselective angiography revealed venograft patency (**Figure 3**). Eighteen months after surgery, the dog was without detectable abnormalities on clinical examination and continued to maintain normal appetite and activity level. The dog had persistent hypoalbuminemia (2.6 g/dL).

Canine thymomas are lymphoepithelial tumors of thymic epithelial cells with lymphocyte populations of varying density.[3] An autoimmune paraneoplastic syndrome of myasthenia gravis, polymyositis, and concurrent nonthymic tumors occurs in 48% to 67% of cases.[4-7] The tumors are classified as benign if well encapsulated within the thymic capsule or malignant if local invasion of the surrounding tissues has occurred.[1,8] Malignant thymomas can metastasize, with reported sites of metastases that include the pericardium, local lymph nodes, lung, diaphragm, liver, spleen, kidney, and bone.[4,9] Definitive diagnosis of thymoma requires biopsy of the mediastinal mass. Exclusion of thoracic lymphoma as a differential diagnosis is essential prior to surgical exploration and attempted resection. Clinical signs in dogs with thymomas are variable and related to a space-occupying cranial mediastinal mass or manifestations of the paraneoplastic syndrome. Edema of the face, neck, and forelimbs with ocular and jugular vein distention is termed cranial vena cava syndrome and is well documented in dogs and humans with thymoma and intrathoracic compression, invasion, or thrombosis of the cranial vena cava.[4,5,10-14] This is a critical condition in humans, often resulting in laryngeal edema, airway obstruction, cerebral venous hypertension, and cyanosis.[13]

Induced hypothermia under anesthesia is routinely used in some vascular and oncologic surgical procedures to diminish potential surgical morbidity. The reduction in body and brain temperature decreases the metabolic rate of brain cells and decreases their oxygen consumption. This is beneficial if hemorrhage during the procedure causes hypovolemia and relative hypoxemia. When carefully performed and closely monitored, the hypothermic procedure has minimal drawbacks.

Successful excision of a malignant thymoma with extension into the vena cava has been accomplished with local venotomy and thrombectomy, with primary repair of the vena cava wall.[12] In the dog of this report, the cranial vena cava was not patent after resection of the tumor, thrombectomy, and reconstruction of the vessel wall. In research dogs, ligation of the cranial vena cava commonly results in development of chylothorax.[15] Therefore, because the dog's clinical signs were caused by obstruction of the vena cava and because of the adverse effects observed in dogs after ligation of the vena cava, an intraoperative decision was made to reconstruct the vessel. Several materials have been used for reconstruction of the cranial vena cava in research dogs, including autogenous external

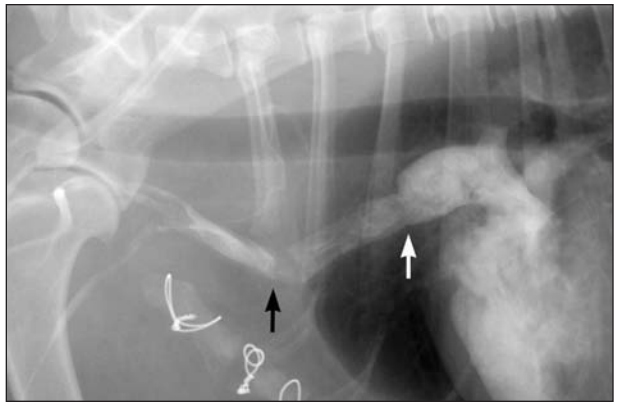

Figure 3—Positive contrast radiographic view of the thorax of the dog of Figure 1 four months after surgery. Notice sites of the cranial (black arrow) and caudal anastomoses (white arrow) formed by use of a jugular vein graft. Continued patency of the graft is evident.

jugular vein,[16] pericardium,[17] autogenous small intestinal submucosa,[18,19] spiral-supported expanded polytetrafluoroethylene,[16,20,21] and fluorocarbon resin.[22] To the authors' knowledge, the only clinical report[12] of attempted vascular reconstruction of the cranial vena cava in a dog involved the insertion of a polytetrafluoroethylene vascular prosthesis, which subsequently became thrombosed.

Successful vascular graft reconstruction of the superior vena cava in humans after enbloc resection of a thymoma has been performed for more than 25 years,[23,24] by use of graft materials that include umbilical vein,[25] saphenous vein,[25] composite spiral saphenous vein,[24] autogenous pericardium,[25,26] externally reinforced polytetrafluoroethylene,[25] and external jugular vein.[27,28] Surgical results were good in most patients, with complete tumor resection often possible, and long-term graft patency was achieved. Graft thrombosis was the major cause of graft failure, but the occurrence rate was low.[23,24,29] In the dog of this report, a jugular venograft was used to reconstruct the cranial vena cava. The jugular vein is readily harvested, and loss of 1 jugular vein is well tolerated in dogs.[30] In research dogs, spiral jugular vein grafts have been used for caudal vena caval reconstruction.[31-33] Clinically, canine jugular vein has also been used successfully as a portocaval venograft in the surgical management of intrahepatic portosystemic shunts.[30,34] The use of silk for vascular anastomosis is controversial. Waxed silk was used because it has excellent handling and knotting characteristics and does not purse-string the vessel lumen at the anastamosis site during placement. Reported problems with silk sutures include thrombogenesis, induction of inflammatory reactions, and fiber shedding from its loose multifilament structure.

The development of localized postoperative subcutaneous edema indicated that the underlying cause was focal in nature. It is possible that the venograft did not allow maximal venous return in the initial period after surgery, leading to an increase in capillary hydrostatic pressure in the tissues of the head and forelimbs. In addition to this, the dog received a large volume of crystalloids and colloids during and immediately after surgery. This would further exacerbate the increased hydrostatic pressure in those capillary beds, leading to extravasation of fluid and development of interstitial edema. While the dog remained recumbent, the normal augmentation of venous return in response to movement and gravity was lost. In the days after surgery, fluid balance was returned to normal and the venous return from the affected areas may have increased because of adaptation of the graft as well as the benefits of gentle exercise. The dog was treated with heparinoid compounds after surgery to prevent thrombosis, which is an important complication of vascular surgery.[35] In research dogs, enoxaparin, a low-molecular-weight heparin, is safe and effective in the prevention and treatment of venous thromboembolism.[36,37] The major advantages of enoxaparin over unfractionated heparin include longer half-life, increased bioavailability, and no requirement for monitoring of bleeding time.[38] Enoxaparin is effective in the prevention and treatment of postoperative thrombosis in human clinical trials.[39] Experimentally, it prevents repetitive platelet-dependent thrombus formation in dogs.[40]

Renal biopsy was performed to better characterize the protein-losing nephropathy and detect glomerulonephritis. The prognosis for glomerulonephritis is variable, whereas that of amyloidosis is typically considered to be poor.[41] Glomerulonephritis has been associated with multiple etiologies including infectious, neoplastic, and inflammatory conditions; neoplasia has been cited in approximately 20% to 40% of cases.[42-44] We believe that the thymoma was the cause of the glomerulonephritis, either because of direct antigenic stimulation or as a paraneoplastic process. The ideal treatment of glomerulonephritis is identification and elimination of the underlying source of antigenic stimulation.[45] When this is not possible, treatment may involve the use of antithromboembolic drugs, dietary sodium and protein restriction, **angiotensin-converting enzyme inhibitors (ACEIs)**, and immunosuppressive therapy.[45] The ACEIs reduce proteinuria and delay the onset or progression of azotemia in dogs with naturally occurring glomerulonephritis, even when they are normotensive initially.[46] The initiation of ACEI therapy in this dog prior to referral may have helped minimize the degree of proteinuria and reduced further risk of tubulo-interstitial damage caused by the proteinuria. The improvement in the proteinuria after the thymoma was removed supported the supposition that the tumor was related to the glomerulonephritis. The degree of proteinuria may not have been sufficient to explain the degree of hypoalbuminemia initially observed; therefore, it is not that surprising that resolution of proteinuria did not lead to complete resolution of hypoalbuminemia. Additional causes of this dog's hypoalbuminemia, however, were not identified.

In the dog of this report, complete tumor resection combined with an autogenous external jugular venograft provided long-term treatment for an invasive malignant thymoma with associated cranial vena cava syndrome. To the authors' knowledge, there is a single report[12] of successful surgical correction of an invasive thymoma with cranial vena caval syndrome in a dog. A primary resection of the affected wall of the cranial vena cava at the site of tumor entry into the vessel wall was followed by primary repair of the vessel in a single longitudinal layer. Substantial compromise of the vessel width was not observed. Use of autogenous venografting for clinical cases in which invasion of the vena cava is not amenable to venotomy and primary vessel reconstruction may allow primary resection of some previously unresectable tumors.

[a]Canine k/d, Hill's Pet Nutrition, Topeka, Kan.
[b]Enacard, Merck AgVet Division, Merck & Co Inc, Rahway, NJ.
[c]Conray 400, Mallinckrodt Inc, St Louis, Mo.
[d]Temno biopsy system, 18-gauge X 9 cm, Ref T189, Allegiance Healthcare Corp, McGaw Park, Ill.
[e]3-0 silk (1.5 metric), 30" (75 cm), Taper 17-mm circle RB-1 needle, K871, Ethicon, Somerville, NJ.
[f]Bone wax 2.5 g W31G, Ethicon, Somerville, NJ.
[g]4-0 silk (1.5 metric), 30" (75 cm), Taper 17-mm circle RB-1 needle, K871, Ethicon, Somerville, NJ.
[h]Surgical 3.5X EF telescope, 22 degree, Designs for Vision Inc, Ronkonkoma, NY.

[i]Argyle trocar thoracic catheter 20Fr (6.7 mm), 16" (41 cm), Ref 8888561043, Kendall Tyco, Mansfield, Mass.
[j]Hetastarch, Abbott Laboratories, North Chicago, Ill.
[k]Metoclopramide injection, Abbott Laboratories, North Chicago, Ill.
[l]Heparin sodium injection, Elkins-Sinn Inc, Cherry Hill, NJ.
[m]Isovue-200, Bracco Diagnostics Inc, Princeton, NJ.
[n]Lovenox (Enoxaparin sodium) injection, Aventis Pharmaceuticals Products Inc, Bridgewater, NJ.

References

1. Day MJ. Review of thymic pathology in 30 cats and 36 dogs. *J Small Anim Pract* 1997;38:393–403.
2. Morris TA, Marsh JJ, Konopka R, et al. Anti-thrombotic efficacies of enoxaparin, dalteparin, and unfractionated heparin in venous thrombo-embolism. *Thromb Res* 2000;100:185–194.
3. Parker GA, Casey HW. Thymomas in domestic animals. *Vet Pathol* 1976;13:353–364.
4. Bellah JR, Stiff ME, Russell RG. Thymoma in the dog: two case reports and review of 20 additional cases. *J Am Vet Med Assoc* 1983;183:306–311.
5. Aronsohn MG, Schunk KL, Carpenter JL, et al. Clinical and pathologic features of thymoma in 15 dogs. *J Am Vet Med Assoc* 1984;184:1355–1362.
6. Atwater SW, Powers BE, Park RD, et al. Thymoma in dogs: 23 cases (1980–1991). *J Am Vet Med Assoc* 1994;205:1007–1013.
7. Lainesse MF, Taylor SM, Myers SL, et al. Focal myasthenia gravis as a paraneoplastic syndrome of canine thymoma: improvement following thymectomy. *J Am Anim Hosp Assoc* 1996;32:111–117.
8. Aronsohn M. Canine thymoma. *Vet Clin North Am Small Anim Pract* 1985;15:755–767.
9. Robinson M. Malignant thymoma with metastases in a dog. *Vet Pathol* 1974;11:172–180.
10. Palmer KG, King LG, Van Winkle TJ. Clinical manifestations and associated disease syndromes in dogs with cranial vena cava thrombosis: 17 cases (1989–1996). *J Am Vet Med Assoc* 1998; 213:220–224.
11. Peaston AE, Church DB, Allen GS, et al. Combined chylothorax, chylopericardium, and cranial vena cava syndrome in a dog with thymoma. *J Am Vet Med Assoc* 1990;197:1354–1356.
12. Hunt GB, Churcher RK, Church DB, et al. Excision of a locally invasive thymoma causing cranial vena caval syndrome in a dog. *J Am Vet Med Assoc* 1997;210:1628–1630.
13. Baker GL, Barnes HJ. Superior vena cava syndrome: etiology, diagnosis, and treatment. *Am J Crit Care* 1992;1:54–64.
14. Kamikubo Y, Shiiya N, Kubota S, et al. Thymic carcinoma with tumor thrombus into the superior vena cava. *Jpn J Thorac Cardiovasc Surg* 2001;49:327–329.
15. Fossum TW, Birchard SJ. Lymphangiographic evaluation of experimentally induced chylothorax after ligation of the cranial vena cava in dogs. *Am J Vet Res* 1986;47:967–971.
16. Fiore AC, Brown JW, Cromartie RS, et al. Prosthetic replacement for the thoracic vena cava: an experimental study. *J Thorac Cardiovasc Surg* 1982;84:560–568.
17. Brais M, Bertranou E, Brassard A, et al. Effect of dextran on patency of pericardial tubular graft of the superior vena cava in the dog. *J Thorac Cardiovasc Surg* 1973;65:296–303.
18. Lantz GC, Badylak SF, Coffey AC, et al. Small intestinal submucosa as a superior vena cava graft in the dog. *J Surg Res* 1992;53: 175–181.
19. Lantz GC, Badylak SF, Hiles MC, et al. Small intestinal submucosa as a vascular graft: a review. *J Invest Surg* 1993;6:297–310.
20. Masuda H, Ogata T, Kikuchi K, et al. Longevity of expanded polytetrafluoroethylene grafts for superior vena cava. *Ann Thorac Surg* 1989;48:376–380.
21. Esato K, Shintani K, Yasutake S, et al. Experimental replacement of vena cava with expanded polytetrafluoroethylene graft. *Int Surg* 1981;66:227–232.
22. Hasegawa T, Hasegawa S, Fukushima K, et al. Prosthetic replacement of the superior vena cava treated with antiplatelet agents. *Surgery* 1987;102:498–506.
23. Jeanfaivre T, Pegis JD, Enon B, et al. Surgical vein reconstruction in patients with tumor invasiveness of the superior caval system. Retrospective study of 7 cases. *J Chir (Paris)* 1996;133:61–64.
24. Doty JR, Flores JH, Doty DB. Superior vena cava obstruction: bypass using spiral vein graft. *Ann Thorac Surg* 1999;67:1111–1116.
25. Larsson S, Lepore V. Technical options in reconstruction of large mediastinal veins. *Surgery* 1992;111:311–317.
26. Piccione W Jr, Faber LP, Warren WH. Superior vena caval reconstruction using autologous pericardium. *Ann Thorac Surg* 1990; 50:417–419.
27. Doty DB, Doty JR, Jones KW. Bypass of superior vena cava. Fifteen years' experience with spiral vein graft for obstruction of superior vena cava caused by benign disease. *J Thorac Cardiovasc Surg* 1990;99:889–896.
28. Li YZ. Surgical treatment of obstructed superior vena cava. *Zhonghua Wai Ke Za Zhi* 1992;30:142–143.
29. Yagi K, Hirata T, Fukuse T, et al. Surgical treatment for invasive thymoma, especially when the superior vena cava is invaded. *Ann Thorac Surg* 1996;61:521–524.
30. Kyles AE, Gregory CR, Jackson J, et al. Evaluation of a portocaval venograft and ameroid ring for the occlusion of intrahepatic portocaval shunts in dogs. *Vet Surg* 2001;30:161–169.
31. Gloviczki P, Pairolero PC, Toomey BJ, et al. Reconstruction of large veins for nonmalignant venous occlusive disease. *J Vasc Surg* 1992;16:750–761.
32. Gloviczki P, Hollier LH, Dewanjee MK, et al. Experimental replacement of the inferior vena cava: factors affecting patency. *Surgery* 1984;95:657–666.
33. Alavaikko A. Spiral autogenous venous graft in the replacement of large vessels. An experimental study with special reference to replacement of the inferior vena cava. *Acta Chir Scand Suppl* 1988;542:1–42.
34. White RN, Trower ND, McEvoy FJ, et al. A method for controlling portal pressure after attenuation of intrahepatic portacaval shunts. *Vet Surg* 1996;25:407–413.
35. Gopalan PD, Burrows RC. Critical care of the vascular surgery patient. *Crit Care Clin* 2003;19:109–125.
36. Ignasiak DP, McClanahan TB, Bousley RE, et al. Effects of intravenous enoxaparin and intravenous inogatran in an electrolytic injury model of venous thrombosis in the dog. *J Thromb Thrombolysis* 1998;6:199–206.
37. McClanahan TB, Hicks GW, Morrison AL, et al. The antithrombotic effects of CI-1031 (ZK-807834) and enoxaparin in a canine electrolytic injury model of arterial and venous thrombosis. *Eur J Pharmacol* 2001;432:187–194.
38. Hull RD, Pineo GF. Therapeutic use of low-molecular-weight heparins. *Haemostasis* 1993;23(suppl 1):2–9.
39. Haas S. The role of low-molecular-weight heparins in the prevention of venous thrombosis in surgery with special reference to enoxaparin. *Haemostasis* 1996;26(suppl 2):39–48.
40. Leadley RJ Jr, Kasiewski CJ, Bostwick JS, et al. Inhibition of repetitive thrombus formation in the stenosed canine coronary artery by enoxaparin, but not by unfractionated heparin. *Arterioscler Thromb Vasc Biol* 1998;18:908–914.
41. Grauer GF, DiBartola SP. Glomerular disease In: Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine*. 5th ed. Phildelphia: WB Saunders Co, 2000;1662–1678.
42. Cook AK, Cowgill LD. Clinical and pathological features of protein-losing glomerular disease in the dog: a review of 137 cases (1985–1992). *J Am Anim Hosp Assoc* 1996;32:313–322.
43. DiBartola SP, Tarr MJ, Parker AT, et al. Clinicopathologic findings in dogs with renal amyloidosis: 59 cases (1976–1986). *J Am Vet Med Assoc* 1989;195:358–364.
44. Grant DC, Forrester SD. Glomerulonephritis in dogs and cats: glomerular function, pathophysiology, and clinical signs. *Compend Contin Educ Pract Vet* 2001;23:739–746.
45. Grauer GF. Glomerulonephritis. *Semin Vet Med Surg (Small Anim)* 1992;7:187–197.
46. Grauer GG, Greco DS, Getzy DM, et al. Effects of enalapril versus placebo as a treatment for canine idiopathic glomerulonephritis. *J Vet Intern Med* 2000;14:526–533.