Surgical treatment of a pulmonary artery vascular hamartoma in a dog

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Case Description—A 6-year-old Siberian Husky–mix dog was examined for episodes of collapse.

Clinical Findings—Physical examination, echocardiography, abdominal ultrasonography, ECG, and thoracic computed tomography with contrast were performed and revealed a 2.5 X 2.3 X 2.0-cm mass over the pulmonic valve leaflets, resulting in moderate pulmonic stenosis. Other abnormal findings included systemic hypertension, right bundle branch block, proteinuria, and a urinary bladder mass.

Treatment and Outcome—Pulmonary arteriotomy was performed under inflow occlusion, and the mass was resected with trancesophageal echocardiographic guidance and direct visualization. Results of histologic examination of the mass revealed a vascular hamartoma. Sequential follow-up examinations and telephone contacts (at 0.5, 5, and 15 months after surgery) revealed that the patient had been free from episodes of collapse since surgery. No regrowth of the mass was noted on follow-up echocardiograms, and the pulmonic stenosis had resolved, although mild to moderate pulmonary insufficiency later developed. The bladder mass was excised 15 months after the first surgery when hematuria developed, and results of histologic examination of this mass revealed a vascular hamartoma. The dog was eventually euthanized 31 months after the initial surgery for reasons that could not be directly linked to any recurrence of the pulmonary artery mass.

Clinical Relevance—Hamartomas are benign tumors that can be located in various tissues, including large arteries. Computed tomography was helpful in predicting the resectability of the intracardiac mass in this dog. Treatment with arteriotomy under inflow occlusion and mild hypothermia resulted in a favorable outcome. (J Am Vet Med Assoc 2012;240:858–862)

Abbreviations

PA Pulmonary artery
RVOT Right ventricular outflow tract

A 6-year-old 32.5-kg (71.5-lb) castrated male Siberian Husky–mix dog with a history of 2 exercise-induced collapsing episodes was referred to the Soft Tissue Surgery Service of the College of Veterinary Medicine, North Carolina State University, for surgical evaluation of a mass in the vicinity of the PA valve. Previously, as determined on the basis of a physical examination at a cardiology specialty practice, the dog was found to be bright, alert, overweight (body condition score, 7/9; normal range, 4/9 to 5/9), and panting heavily. The heart rate was 124 beats/min, and a grade 5/6 holosystolic ejection quality heart murmur was heard most audibly at the left heart base; the second heart sound was split. An ECG recorded at admission revealed right bundle branch block; the systolic blood pressure (170 mm Hg; reference range, 110 to 140 mm Hg) was repeatedly measured by use of Doppler methodology with a No. 4 cuff on the left hind limb with the dog in right lateral recumbency. A 2-D and Doppler echocardiogram obtained prior to surgical referral revealed a 2.5 X 2.3 X 2.0-cm ovoid, heterogeneous mass moving back and forth with the pulmonic valve between the RVOT and the PA, partially obstructing the PA (Figure 1) and generating a peak instantaneous pressure gradient of approximately 70 mm Hg across the lesion. The pulmonic valve leaflets were reported to be distorted. Moderate right ventricular enlargement and mild interventricular septal flattening during diastole were considered by the referral cardiologist to be commensurate with the degree of pulmonic stenosis caused by the mass. There was minimal infundibular hypertrophy noted, and the remainder of the echocardiogram was reported to be unremarkable.

On arrival at the hospital at North Carolina State University, a CBC, serum biochemistry panel, urinalysis, and abdominal ultrasonographic examination were performed. The CBC and serum biochemical analysis revealed no abnormalities. The urinalysis revealed 3+ proteinuria, and the urine protein-to-creatinine ratio was elevated (1.69; reference ratio < 1). Abdominal ultrasonography revealed an apical polypoid urinary bladder mass.

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Helical computed tomographic examination of the chest was performed to further characterize the intracardiac mass. This examination was performed under general anesthesia with continuous monitoring of vital signs (heart rate, respiratory rate, systolic arterial pressure, mean arterial pressure, diastolic arterial pressure, oxygen saturation as measured with pulse oximetry, and end tidal partial pressure of CO₂). Contiguous 1-mm-thick transverse images were obtained with a 16-slice multidetector device with a pitch of 0.625°. A single injection of nonionic iodinated contrast medium was administered IV (770 mg of iodine/kg [350 mg of iodine/lb] via a catheter that had been placed in a cephalic vein, and contrast angiographic scans were obtained immediately as well as 30 and 60 seconds after contrast injection. Images were reconstructed with a medium frequency algorithm for soft tissues with a reconstruction interval of 1 mm. The images showed a rounded filling defect with soft tissue attenuation located in the RVOT at the level of the pulmonic valve (Figure 2). This filling defect appeared bilobed on some sequences, possibly attributed to valve motion artifact. A cranial mediastinal mass measuring 22.2 mm in height was also found, most consistent with cranial mediastinal lymphadenopathy.

The intracardiac mass was thought to be resectable, and the owner consented to surgery. The next morning, the dog was routinely prepared for a lateral thoracotomy. The dog was premedicated with acepromazine maleate (0.01 mg/kg [0.005 mg/lb], IV) and buprenorphine hydrochloride (0.01 mg/kg, IV) and anesthesia was induced with propofol (3.6 mg/kg [1.6 mg/lb], IV). An endotracheal tube was placed, anesthesia was maintained with isoflurane (1% to 2%) in oxygen, and the dog was allowed to breathe spontaneously. Vital signs (heart rate, respiratory rate, systolic arterial pressure, mean arterial pressure, diastolic arterial pressure, oxygen saturation, end tidal partial pressure of CO₂, and rectal temperature) were monitored continuously throughout anesthesia. Mild hypothermia (rectal temperature, 34.0° to 34.4°C [93° to 94°F]) was induced after anesthetic induction and before surgery (by passive cooling and spraying externally with alcohol) as well as during surgery (by lavaging the thoracic cavity with cold sterile saline [0.9% NaCl] solution; bags stored at 4°C [39.2°F]). Following routine left thoracotomy at the fourth intercostal space, the cranial vena cava and the azygous vein were located and umbilical tape was loosely placed around them and guided through cut portions of a 14F red rubber catheter to act as Rummel tourniquets. An additional left sixth intercostal space thoracotomy was then performed to isolate the caudal vena cava, and a tourniquet was placed around it in a similar manner. Incisions into the pericardium were made from the fourth intercostal space, ventral and dorsal to the phrenic nerve. The location of the mass was confirmed with transesophageal echocardiography (Figure 3). Four stay sutures (4-0 polypropylene) were placed left, right, dorsal, and ventral to the pulmonary arterial incision. After inflow occlusion, the PA was incised between the preplaced stay sutures. The mass was found to have a relatively narrow stalk and was sharply excised from the lateral wall of the PA just distal to the lateral leaflet (supravalvular location). Complete exci-
sion of the mass was documented with transesophageal echocardiography. After excision of the mass, a Satinsky clamp was placed adjacent to the incision and the Rummel tourniquets were released. Total inflow occlusion time was 2.5 minutes. The pulmonary arteriotomy was closed with a combination of a simple continuous suture (4-0 polypropylene) and simple interrupted mattress sutures on pledgets. Gentle pressure was applied on the arteriotomy site as the Satinsky clamp was removed. The sternal lymph node was then aspirated under direct visualization with a 22-gauge needle and syringe. Because of the presence of multiple vessels surrounding it, lymph node removal or biopsy was avoided. After confirming the absence of bleeding from the arteriotomy site, a thoracic tube was placed and the thorax was closed in a routine manner.

Mild systemic hypertension, as previously identified preoperatively, persisted during the postoperative period, and treatment with an angiotensin-converting enzyme inhibitor was initiated. The dog made an otherwise uneventful recovery in the intensive care unit, and the thoracic tube was removed 20 hours after surgery. The dog was discharged 48 hours after surgery with the following medications: enalapril (5 mg, PO, q 12 h), tramadol (50 mg, PO, 2 to 3 times daily as needed for pain), and a fentanyl patch (100 µg) applied transdermally for 5 days.

On histologic examination, the unencapsulated mass was composed of numerous variably sized, blood-containing vascular channels that were irregularly arranged in collagenous stroma (Figure 4). Several large, 5-mm-diameter blood vessels resembling nonneoplastic arteries or veins were lined by normal-appearing, flat endothelial cells supported by a thin to thick fibrous wall containing smooth muscle cells. Interspersed small vessels resembled capillaries and small veins. Occasionally, small primitive vessels were lined by endothelial cells with moderately enlarged, hyperchromatic nuclei with prominent nucleoli, exhibiting minimal to mild cellular atypia. Plump spindle cells, arranged in small clusters and primitive small channels, were bordered by or interspersed between large vessels. These cells often contained a clear cytoplasmic vacuole and infrequent mitotic figures (< 1/10 hpf). The lesion was consistent with a histopathologic diagnosis of chronic vascular hamartoma on the basis of marked hemosiderin accumulation from repeat hemorrhage and areas of prominent sclerosis. Cytologic examination of the mediastinal lymph node aspirate was poorly cellular and failed to identify notable abnormalities.

At a follow-up examination performed 2 weeks after surgery, the owner reported that no more collapsing episodes had occurred, and as determined on the basis of a physical examination, the dog was noted to be bright and alert. The cardiopulmonary examination indicated a heart rate of 100 beats/min, with a soft grade 2/6 systolic ejection heart murmur heard best at the left heart base; the respiratory rate was 30 breaths/min, and the lung sounds were normal. An echocardiogram showed resolution of the previously noted pulmonic stenosis, with persistent mild to moderate right heart enlargement and no pulmonic valve mass. The lateral leaflet of the pulmonic valve was difficult to visualize, but the rest of the valve appeared to move normally. An ECG recorded during echocardiography revealed persistent right bundle branch block. Urinalysis showed a reduction in proteinuria (urine protein-to-creatinine ratio of 1; reference limit < 1). The systemic arterial blood pressure as measured with Doppler methodology was normal (135 mm Hg); a serum biochemical analysis revealed no abnormalities. A CBC was not performed at that time.

On subsequent recheck examinations 5 and 15 months after surgery, echocardiography continued to show moderate right heart enlargement, but the previously noted interventricular septal flattening had resolved and the pulmonary outflow velocity was normal. No regrowth of the mass was noted in the area of the pulmonary arteriotomy. Mild to moderate low-velocity pulmonic insufficiency was present. The systemic arterial blood pressure was slightly elevated (150 mm Hg), but the dog appeared to be nervous during the examinations. The dog's owner did not note any further collapsing episodes and had elected to forego further evaluation and treatment for the bladder mass; however, the dog had developed hematuria by the 15-month postoperative visit. The owner consented to further treatment at that time, and a cystotomy was performed to explore the bladder mass. A pedunculated mass was found at the apex of the bladder, and histopathologic results were consistent with a benign vascular hamartoma, similar to the pulmonic valve mass. Immediately after surgery, the dog had an episode of mild azotemia, with a creatinine value of 2.2 mg/dL (reference range, 0.7 to 1.6 mg/dL) and BUN
value of 32 mg/dL (reference range, 6 to 25 mg/dL). The values were normal by postoperative day 2 (creatinine, 1.7 mg/dL; BUN, 14 mg/dL), and the dog was discharged without further complications. The dog was then examined by the referring veterinarian for its annual visit 29 months after the initial surgery (14 months after the second surgery), and although the owners did not report any change in its amount of activity, physical examination results revealed marked weight loss (body weight, 25 kg [55 lb]), no heart murmur, arrhythmias, or hematuria was noted. A CBC, serum biochemical analysis, and urine analysis performed at this time did not reveal any abnormalities. No further imaging was performed, and medical treatment (ie, increase calorie intake) was refused because the dog was emaciated (44 lb). Two years after surgery, the dog was nonambulatory and had a urine-soaked abdomen and an irregular heart rhythm was noted, but no murmur or hematuria was identified. At the request of the owner, the dog was euthanized and a necropsy was not performed.

Discussion

In the present report, a dog underwent successful surgical treatment of a PA valvular hamartoma under in-flow occlusion with an excellent outcome (survival > 2 years after surgery). The patient was euthanized > 2.5 years after surgery for reasons presumably unrelated to a regrowth of the mass in the PA or bladder but that could have been related to recurrence or metastasis, although we speculate that this was unlikely. Other conditions that might have been the cause of death were chronic renal disease, age (the dog was 11 years old when euthanized) could potentially explain the clinical signs more readily than could recurrence of a hamartoma, such as canine cognitive disorder, development of other neoplasia, or brain disorders. However, no formal conclusion can be drawn because a necropsy was not performed.

Hamartoma of the pulmonary infundibulum has been described in a human patient, but to our knowledge, this is the first report of hamartoma in the PA of a dog, although it has been previously found in other vessels. Other locations of hamartoma described in the veterinary literature (restricted to companion animals) include the liver, nephroneoplastic structures (spinal cords, brain, and cranial nerves), spleen, skin, oral cavity, heart, genitalia, kidney, lung, intestines, and genitourinary tract, skin, breast, thyroid gland, and nervous and musculoskeletal systems) and predispose to cancer development in those affected areas.

The mass in the patient in the present report caused moderate supravalvular pulmonic stenosis. Scansen et al described acquired pulmonic stenosis in 4 dogs, one of which was caused by an intravascular mass (fibrosarcoma) located in the main PA. The use of a patch to increase the size of the RVOT following mass resection was described because the resection seemed to have narrowed it. In our patient, we did not think that the resection site would substantially narrow the RVOT; therefore, a patch was unnecessary.

The systemic hypertension noted in the dog in the present report could be related to undiagnosed glomerular disease (as supported by an increase in the urine protein-to-creatinine ratio at initial examination). However, because no measurement of glomerular filtration rate or renal biopsies were performed, the exact cause of the systemic hypertension in this patient remains speculative.

a. Parks Medical Electronics Inc, Aloha, Ore.
b. Somatom Sensation 16, Siemens AG, Forchheim, Germany.
c. jCode, Omnipaque 350 mg/mL, General Electric Healthcare, Waukesha, Wis.
d. 14F red rubber open urethral tray, Covidien Healthcare, Boston, Mass.
e. iE33, Royal Philips Electronics, Amsterdam, The Netherlands.
f. Prolene, Ethicon Inc, Somerville, NJ.
g. Dacron pledgets, Boston Scientific Inc, Waynetown, NJ.
h. iohexol, Omnipaque 350 mg/mL, General Electric Healthcare, Waukesha, Wis.
i. Enalapril maleate, Wockhardt Ltd, Mumbai, India.
j. Amneal Pharmaceuticals of NY, Hauppauge, NY.
k. Fentanyl transdermal systems, Watson Pharmaceuticals Inc, Corona, Calif.
References


From this month’s *AJVR*

**Effect of optical defocus on performance of dogs involved in field trial competition**

Ron Ofri et al

**Objective**—To measure the effect of induced myopia on field trial performance in dogs.

**Animals**—7 Labrador Retrievers and 1 Chesapeake Bay Retriever trained in field trial competition.

**Procedures**—Dogs were commanded to retrieve targets at 137.2 m (150 yards). Each dog participated in 3 trials while their eyes were fitted with 0- (plano), +1.50-, or +3.00-D contact lenses, applied in random order. Retrieval times were measured objectively, and dog performances were evaluated subjectively by masked judges.

**Results**—Retrieval times were significantly faster with plano lenses than with +1.50- or +3.00-D lenses, but there were no significant differences in times between +1.50- and +3.00-D lenses. Masked judges assigned the best performance scores to dogs with plano lenses and the lowest scores to dogs fitted with +3.00-D lenses.

**Conclusions and Clinical Relevance**—Even mild myopic defocusing had a significant negative impact on both the subjective and objective assessments of dogs’ performances. Dogs with demanding visual tasks or signs of visual deterioration should be evaluated retinoscopically to determine the refractive state because they may have ametropia. (*Am J Vet Res* 2012;73:546–550)