Pathology in Practice

In collaboration with the American College of Veterinary Pathologists

Figure 1—Postmortem images of the proximal thoracic aortic intraluminal obstruction (A) and the abdominal aortic intraluminal obstruction imaged between the adrenal glands (B) from an 8.75-year-old castrated male West Highland White Terrier. A—An intraluminal focal, well-demarcated, raised, firm, white, conical mass-like structure (asterisk) is attached to the vascular wall. Also evident is concentric hypertrophy of the left ventricle. B—A large, diffuse, white, pale, firm structure extending through the whole length of the vessel lumen. The adrenal glands (asterisks) are similar in size, and each has a mildly swollen, pale medulla.

History

An 8.75-year-old 9.5-kg castrated male West Highland White Terrier was submitted for postmortem examination. The animal was presented initially with a 4-week clinical history of chronic corneal ulceration in the left eye. On a reevaluation consult 2 weeks after the initial clinical presentation, increased intraocular pressure and elevated systemic blood pressure were detected. Despite medical intervention, the dog worsened, had cardiac arrest, and died.

Clinical and Gross Findings

At first consultation, physical and ophthalmic examinations were performed, and a diagnosis of Spontaneous Chronic Corneal Epithelial Defect affecting the left eye was made. The ulcer was treated with a corneal burring technique, and the animal was reexamined after 2 weeks. Upon reevaluation of the

patient, the left corneal ulcer had healed; however, ophthalmologic examination revealed increased intraocular pressure (33 mm Hg right eye; 25 mm Hg left eye; reference range, 10 to 20 mm Hg), severe bilateral panuveitis, and retinal detachment with small intraretinal hemorrhages. Body temperature was 38.8 °C, oral mucosae were normal, and there was no evidence of pain or abnormalities on palpation of the abdomen or vertebral column. The hematologic and biochemical analyses were within normal limits; however, systemic systolic blood pressure was high (280 mm Hg; reference range, 130 to 160 mm Hg). On serologic qualitative tests (Snap 4Dx Plus test, Idexx laboratories Inc), the dog tested negative for heartworm disease, ehrlichiosis, Lyme disease, and anaplasmosis. The thyroid profile was within normal limits. The animal was admitted for hospitalization and further investigations. The following day, the dog was found to be acutely tachycardic (persistently above 240 bpm), hypertensive, and tachypneic. On ECG, a sinus tachycardia was identified. Despite medical intervention, the dog went into cardiac arrest and died.

On postmortem examination, the proximal thoracic aorta contained a focal intraluminal, well-demarcated, raised, firm, white, conical mass-like structure (30 X 15 X 10 mm) attached to the vascular wall (**Figure 1**). The abdominal aorta contained a long (120 X 15 X 12 mm), white, translucent, and firm multilobulated mass-like structure that extended

Antonia Morey-Matamalas, BVM¹*; Jessica L. Adamany, DVM²; Victor Caudal, DVM²; Davide Corbetta, DVM¹; Ana L. Ortiz, DVM, PhD¹

¹School of Veterinary Medicine and Science, University of Nottingham, Sutton Bonington, England

²Pride Veterinary Centre, Derby, England

*Corresponding author: Dr. Morey-Matamalas
(antonia.moreymatamalas2@nottingham.ac.uk)

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the entire length of the vessel, was attached to the vessel wall, and almost completely filled the lumen. The adrenal glands were similar in size and presented a mildly swollen, pale medulla. The heart exhibited concentric hypertrophy of the left ventricle, with a narrowed ventricular lumen. The left atrioventricular valve exhibited gross changes consistent with mild multifocal endocardiosis. Other macroscopic findings included moderate pulmonary edema and congestion, hepatic congestion, mild chronic interstitial nephritis, and splenic nodular hyperplasia. No other major macroscopic findings were identified during the autopsy.

Formulate differential diagnoses, then continue reading.

Histopathologic Findings

Samples of the mass affecting the proximal thoracic and abdominal aorta were collected, fixed in neutral-buffered 10% formalin, and routinely processed for histologic examination.

The aortic mass consisted of an unencapsulated, densely cellular, well-demarcated neoplasm composed of lobules of haphazardly arranged large, round to spindle neoplastic cells (chondrocytes), surrounded by abundant, variably basophilic, chondromatous matrix, separated by strands of vascularized fibrous strands (**Figure 2**). The mass arose from the tunica intima and filled the lumen of the aorta (**Figure 3**). Neoplastic cells were individualized or congregated in lacunae within the matrix, had discrete cell borders, and had a moderate amount of finely granular eosinophilic to basophilic cytoplasm. Nuclei were round to oval and had up to 1 magenta nucleolus and finely stippled to hyperchromatic chromatin. Mitoses were 1 in 10 hpfs. Anisocytosis and anisokaryosis were mild with few binucleated cells (**Figure 2**). Multifocally within the neoplasm, there were low numbers of small mature lymphocytes and plasma cells admixed with hemorrhages, minimal fibrin, and small amount of basophilic amorphous material (mineral). The tunica media and adventitia multifocally contained small numbers of previously described inflammatory cells admixed with edema, low numbers of hemosiderin-laden macrophages and hemorrhages, and mildly increased numbers of reactive fibroblasts. Within adjacent collateral small arterioles, there was moderate thickening of the tunica media (media hypertrophy), the lumen was reduced, and the intimal endothelium was reactive, exhibiting hypertrophy.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis: aortic chondrosarcoma.

Case summary: primary aortic chondrosarcoma affecting cranial and caudal (abdominal) portions of the aorta in a dog with hypertension.

Figure 2—Photomicrographs of sections of the aortic mass from the dog described in Figure 1. A—The neoplasm is composed of haphazardly arranged large, round to spindle well-differentiated (asterisk) and poorly differentiated (double asterisks) neoplastic cells (chondrocytes) surrounded by irregular islands and thick trabeculae of abundant, variably basophilic, chondromatous matrix. H&E stain; bar = 100 µm. B—Higher magnification showing a binucleated neoplastic cell. H&E stain; bar = 50 µm.

Figure 3—Photomicrograph of section of the aortic mass in the dog described in Figure 1. The neoplasm arises from the tunica intima of the aorta and fills the aortic lumen. H&E stain; bar = 500 µm.

Comments

Chondrosarcoma (CSA) is the second most common primary bone tumor in dogs and humans. In dogs, it accounts for approximately 5% to 10% of all canine primary bone tumors, and, as in humans, it affects flat bones more frequently than long bones.^{1,2} It occurs most often in medium to large breeds and is considered rare in small and giant breeds. Middle-aged to older dogs are most commonly affected, with a mean age ranging from 5.9 to 8.7 years.¹ CSA can also occur in the soft tissue and are referred to as extraskeletal CSA. Documented cases of extraskeletal

CSA include the following: the mammary gland, heart valves, aorta, larynx, trachea, lung, retroperitoneum, urinary bladder, and omentum.^{1,2} Extraskelatal chondrosarcomas involving cardiac chambers or large vessels, such as the aorta, however, are very rare both in humans and animals.^{3–8} Histologically, CSA generally consists of lobules of mesenchymal cells producing variable quantities of disorganized hyaline cartilage or fibrillar matrix, but not osteoid.⁹ In this case, detailed postmortem examination revealed a large mass occluding the cranial and caudal (abdominal) aorta. Histologic examination showed an intimal neoplastic proliferation morphologically compatible with a chondrosarcoma. Given the absence of primary skeletal lesions, a primary abdominal aortic chondrosarcoma was diagnosed.

The clinical presentation reported in the few available case reports of aortic chondrosarcoma in dogs included progressive paresis,^{4,5} ataxia,^{4,6} tetraparesis,^{4,6} and collapse/seizure-like activity.⁷ In 2 cases, dilated cardiomyopathy⁶ and left ventricular concentric hypertrophy⁷ were diagnosed. In the present case, ocular disease (severe bilateral panuveitis and retinal detachment) and systemic hypertension were the main presenting clinical signs.

Clinically recognized hypertension usually occurs in middle-aged to older dogs and cats. Documented or suspected causes of hypertension in dogs and cats include renal disease (especially glomerular), hyperadrenocorticism, hyperthyroidism, pheochromocytoma, diabetes mellitus, hyperaldosteronism, intracranial lesions, and obesity.¹⁰ In this case, no evidence of the abovementioned diseases were present. Histologic examination of both adrenal glands was unremarkable with no evidence of a pheochromocytoma (or any other adrenal neoplasm). The cause of the systemic hypertension was suspected to be the narrowing of the aortic lumen caused by the neoplastic proliferation. In the veterinary literature, pulmonary hypertension was observed in a case of obstructive chondrosarcoma in a dog with concurrent aortic dissection.⁷ In humans, a similar possible mechanism is suggested in a case of primary intimal sarcoma presented as a hypertensive crisis.¹¹ The suggested mechanism is similar to that presented with coarctation of the aorta in humans, which is associated with systemic hypertension through reduced arterial compliance and blunted baroreflex sensitivity.^{10,12} In dogs, ocular fundic changes are common complications associated with systemic hypertension and include bullous to complete retinal detachment, intraretinal edema, and hemorrhage. Moreover, cardiac changes such as left ventricular hypertrophy and left-sided congestive heart failure (rare) are also associated with systemic hypertension.¹⁰ In this case, the retinal and choroidal changes were most likely secondary to the systemic hypertension, likely caused by aortic neoplastic thromboembolism. Bilateral bullous retinal detachments were previously reported in a case of aortic chondrosarcoma in a dog.⁴ The

near complete obstruction of the aortic lumen by the neoplasm, in conjunction with the likely consequent systemic hypertension, was the most likely cause of the compensatory concentric left ventricle hypertrophy. The presence of pulmonary edema and multiorgan congestion secondary to congestive heart failure and inadequate tissue perfusion due to the aortic neoplasm likely caused the death of this patient. Moreover, exercise-induced hypertension has been described in human patients with aortic coarctation and linked to cardiovascular events,¹³ and emotional stress has been shown to increase aortic peak velocity in Boxer dogs.¹⁴ In our case, hospitalization and associated stress could have worsened hypertension and precipitated congestive heart failure.

CSA is considered to have a low metastatic rate (20%), compared to osteosarcoma.² The most common site of metastasis is the lung.^{1,9} Recently, Vinayak et al¹⁵ described an uncommon aggressive variant of CSA in a cat and 7 dogs. The metastatic rate in the reported cases was 63%.¹⁵ No metastases were observed in the present case.

In people, aortic tumors are a malignant pathologic condition with a short survival rate after initial diagnosis. Survival time is further diminished in the presence of clinical factors such as hypertension, fever, back pain, asthenia, and signs of peripheral embolization.¹⁶ In veterinary medicine, the prognosis is not established. In the reported cases, the animals were humanely euthanized shortly after presentation of clinical signs. Regarding treatment, and considering canine CSA as a group independently of the location, aside from surgical resection, a reliable adjuvant chemotherapeutic agent is not currently known.²

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