A 10-year-old 20.5-kg (45.1-lb) spayed female Staffordshire–Bull Terrier crossbred dog was evaluated because of a history of raspy breathing during the preceding week.

On clinical examination, the dog appeared bright and alert and was mildly overweight (body condition score, 6/9), and its rectal temperature (38.2°C [100.76°F]), pulse rate, and respiration rate were within reference limits. On thoracic auscultation, the heart sounds and rhythm were considered normal but a slight inspiratory stridor was noted. No other important abnormalities were detected.

Thoracic radiography revealed a mass effect at the left side of the heart base. Cardiac border effacement suggested that the mass lay within the cranial to mid mediastinum and was in contact with the heart.

Cytologic examination of the prepared smears revealed that the fine-needle aspirate samples were highly cellular with poor cellular preservation; many bare nuclei were scattered in the background (Figure 1). Intact cells were seen singly and in small clusters. These intact cells had pale blue-gray, agranular, mildly to moderately abundant cytoplasm with indistinct borders, appearing to be continuous with the background. Nuclei were round to mildly pleomorphic with stippled chromatin patterns and prominent small round nucleoli. Mild anisokaryosis and variation in the nuclear-to-cytoplasmic ratio were also present. A guarded to poor prognosis for the dog was given to the owner, who elected to euthanize the dog. At necropsy, an irregular, moderately soft gray to pink mass (30 mm in diameter) was identified lateral to the pharynx. The mass was intimately associated with and surrounded the internal and external carotid arteries, displacing the vagosympathetic trunk and hypoglossal nerve. On cut surface, the mass was pink and soft and oozed blood. Two masses of similar size and appearance were identified over the left side of the heart base: one lateral to the origin of the pulmonary artery and the other nestled within connective tissue between the roots of the aorta and pulmonary artery. These masses did not compress the atria or infiltrate cardiac tissues. All of these masses were excised for histologic examination. The pericardial sac was grossly normal, and no other gross abnormalities were detected, although the CNS was not examined.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page →

Figure 1—Photomicrograph of a cytologic preparation from a firm nonmobile 30- to 40-mm-diameter submucosal pharyngeal mass in a 10-year-old Staffordshire–Bull Terrier crossbred dog that was evaluated because of a 1-week history of raspy breathing. Notice the frequent bare nuclei and cells with mildly abundant pale blue cytoplasm and variably distinct borders. The cells have mildly pleomorphic nuclei with stippled chromatin and prominent small nucleoli. Mild anisokaryosis and variation in the nuclear-to-cytoplasmic ratio are also present. Wright-Giemsa stain; bar = 10 µm.
Histopathologic Findings

Histologic examination of sections of the submucosal pharyngeal mass revealed a thinly encapsulated proliferation of monomorphic cells (Figure 2). The cells were polyhedral and arranged in packets with fine collagenous trabeculae. The cells had central round to mildly pleomorphic, hyperchromatic nuclei; finely granular eosinophilic cytoplasm; minimal anisokaryosis; and a low mitotic rate. Occasional giant cell forms were present. The mass was locally invasive and had invaded blood vessels. Histopathologic findings of the heart base masses were similar, but they each had a central focus of cells with marked pleomorphism and many multinucleate and megakaryocytic forms; those cells had hyperchromatism, large pink nucleoli, and a high mitotic rate. The tumor was locally and vaso-invasive (Figure 3). Sections of the submucosal pharyngeal mass and one of the aortic body masses underwent immunohistochemical staining for synaptophysin and chromogranin A. All sections had strong cytoplasmic staining for both synaptophysin and chromogranin A, except 1 heart base mass section, which was positive for synaptophysin but negative for chromogranin A.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis and case summary: carotid and aortic body carcinomas (chemodectomas) in a dog.

Comments

On the basis of the gross findings, differential diagnoses for the pharyngeal mass in the dog of this report included neoplasia, abscess, granuloma, hematoma, branchial cyst, sialocele, or local lymphadenopathy. Radiography revealed a mass effect at the left side of the heart base, and differential diagnoses for this Roentgen sign included heart base tumor; other mediastinal, tracheal, bronchopulmonary, or esophageal tumors (primary or metastatic [eg, ectopic thyroid carcinoma]); lymphadenopathy; abscess; granuloma; hematoma; and pulmonary arterial aneurysm or dilatation (heartworm or congenital left-to-right shunt). As a result of further investigation, a diagnosis of carotid and aortic body carcinomas was made.

The carotid and aortic bodies are chemoreceptors that help to regulate the circulatory and respiratory systems. The carotid bodies are found at the carotid bifurcation, and the aortic bodies are located around the base of the heart.\(^1\,3\) Neoplasms of these organs and other parts of the chemoreceptor system are generally known as chemodectomas or paragangliomas.

Chemodectomas are rare in dogs, with sporadic case reports and case series found in the veterinary medical literature.\(^1\,3\)\(^\text{--}16\) Chemodectomas usually de-
velop in dogs that are 10 to 15 years of age; males seem to be more commonly affected than females, particularly among the brachycephalic breeds, although there might not be a sex predilection associated with carotid body tumors.4,11 Sexual intact female dogs may be less likely to develop aortic body tumors than are spayed female dogs.4 In dogs, aortic body tumors are diagnosed more often than carotid body tumors, by a factor of 5 to 8 in various reports8,10, however, dogs with carotid body tumors frequently have a concomitant aortic body tumor.9

Chemodectomas are generally nonfunctional neoplasms12; however, they are space-occupying lesions and can invade surrounding tissues, metastasize, or become very large (up to 13 cm in diameter).2,10,11 Aortic body tumor space-occupying effects include signs of right-sided congestive heart failure or pericardial effusion due to compression of the atria and venous return to the heart.2,3 Aortic body carcinomas can invade the atria or pericardial sac and may metastasize to the lungs, liver, and other organs or cause spontaneous intracavitary hemorrhage.5,8 Carotid body tumor space-occupying effects include dyspnea, esophageal disease, and circulatory disturbances due to compression of the carotid artery.4,11 Carotid body carcinomas can invade tissues locally, causing neurologic problems such as Horner syndrome and may metastasize to the lungs, bronchial or mediastinal lymph nodes, liver, brain, heart, and other sites.4,11

Aortic body tumors are generally more likely than aortic body tumors to be malignant. The tumors in the dog of the present report were diagnosed as carcinomas because, despite their small size and lack of gross invasion or metastasis, multiple histologic criteria of malignancy were present; in addition, 1 aortic body tumor failed to stain with chromogranin A. Chemodectomas are generally positive for synaptophysin and chromogranin A,4,16,19 and it has been reported that this staining pattern is often lost with increasing malignancy.19,20 It can be difficult to obtain satisfactory cytologic samples from neuroendocrine tissues because of the fragility of their component cells, and some authors have had no success with diagnosis of chemodectomas via examination of fine-needle aspirate specimens.11,16 Nevertheless, fine-needle aspiration yielded a diagnostic specimen in the case described in this report, as it has in others,21 and the procedure should be carefully considered when pursuing diagnosis of suspected carotid body tumors in dogs, particularly when referral for advanced imaging is not available or is declined by the owner.

For dogs with chemodectomas, chemotherapy has been described but was not shown to prolong survival time in 1 study.12 Radiation therapy is of uncertain utility in dogs.5 The mainstay of treatment of chemodectomas is surgery, which can be a challenging prospect. The vascularity of these tumors and their close association to major vessels and nerves make resection difficult, even in the case of benign neoplasms.11,12,16 Postoperative complications are common; in 1 study,11 4 of 10 dogs undergoing carotid body tumor resection died soon after surgery, and survivors often developed Horner syndrome or laryngeal paralysis. However, 5 of those dogs that survived the initial postoperative period had survival times > 12 months.12 It should be noted that surgery may still be of benefit to dogs with unresectable aortic body tumors; pericarctectomy has been shown to dramatically improve survival times of dogs with heart base tumors, regardless of whether they had a pericardial effusion at the time of surgery.12,16

References