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What Is Your Diagnosis?

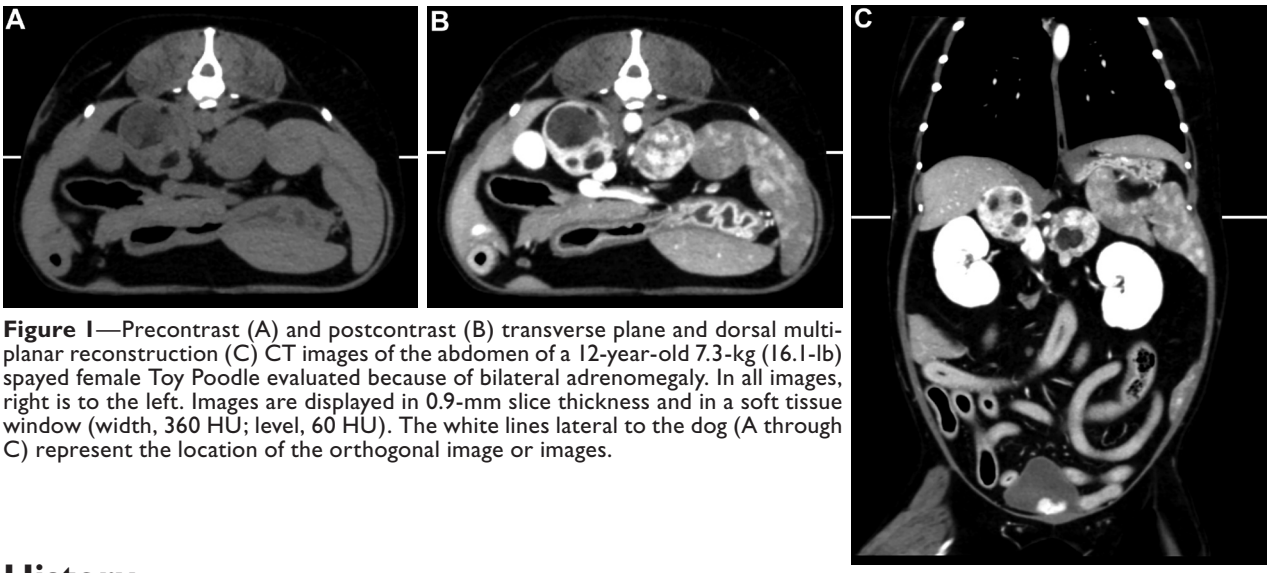

Figure 1—Precontrast (A) and postcontrast (B) transverse plane and dorsal multiplanar reconstruction (C) CT images of the abdomen of a 12-year-old 7.3-kg (16.1-lb) spayed female Toy Poodle evaluated because of bilateral adrenomegaly. In all images, right is to the left. Images are displayed in 0.9-mm slice thickness and in a soft tissue window (width, 360 HU; level, 60 HU). The white lines lateral to the dog (A through C) represent the location of the orthogonal image or images.

History

A 12-year-old 7.3-kg (16.1-lb) spayed female Toy Poodle was referred for evaluation of progressive bilateral adrenomegaly. Adrenomegaly was first noted 13 months earlier, during abdominal ultrasonography performed by the referring veterinarian when investigating polyuria, polydipsia, and high liver enzyme activity in the dog. A gallbladder mucocele was suspected and treated medically with ursodeoxycholic acid (13.8 mg/kg [6.3 mg/lb], PO, q 24 h) for 1 year. Two weeks before referral, the dog underwent follow-up abdominal ultrasonography, and the referring veterinarian noticed that although the abnormalities of the gallbladder were static, the dog's bilateral adrenomegaly had progressed with potential vascular invasion. One week before referral, medical treatment for the gallbladder mucocele was discontinued.

On initial referral examination, the dog was bright and alert and had vital signs within reference limits, but was overweight (body condition score, 7/9) and had a grade 3/6 left systolic heart murmur. In addition, the owners reported that the dog had developed polyphagia at home and panting at rest.

Results of serum biochemical analyses indicated high activity of alkaline phosphatase (1,733 U/L; reference range, 13 to 240 U/L) and alanine aminotransferase (338 U/L; reference range, 18 to 100 U/L) along with high cholesterol concentration (390 mg/dL; reference range, 130 to 354 mg/dL). Results of an ACTH-stimulation test were consistent with hyperadrenocorticism (prestimulation serum cortisol concentration, 4.4 µg/dL [reference range, 1.0 to 5.9 µg/dL]; poststimulation serum cortisol concentration, > 50 µg/dL [reference range, 6.5 to 17.5 µg/dL]). Endogenous ACTH concentration was measured; however, results were equivocal (4.0 pmol/L; reference range, 6.7 to 25.0 pmol/L) and could not rule out a functional adrenal tumor. Therefore, urine metanephrine-to-creatinine and normetanephrine-to-creatinine ratios were determined, but these results were also equivocal (metanephrine-to-creatinine ratio, 165 µg/g [reference range, 16 to 150 µg/g]; normetanephrine-to-creatinine ratio, 308 µg/g [reference range, 37 to 261 µg/g]).

Thoracic and abdominal radiography revealed mild left atrial enlargement and mild diffuse hepatomegaly. Findings on abdominal ultrasonography confirmed the presence of bilateral adrenomegaly, with both adrenal glands having a mass-like appearance and containing multiple hyperechoic nodules that had mild distal acoustic shadowing; however, there was no evidence of vascular invasion. The liver was mildly diffusely enlarged and hyperechoic, and the gallbladder contained nonstructured dependent sludge. The dog underwent general anesthesia for noncontrast CT of the head and abdomen followed by dual-phase CT angiography of the abdomen and venous phase CT of the head. Images were captured with 0.9-mm slice thickness in a soft tissue window (width, 360 HU; level, 60 HU; **Figure 1**).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page →

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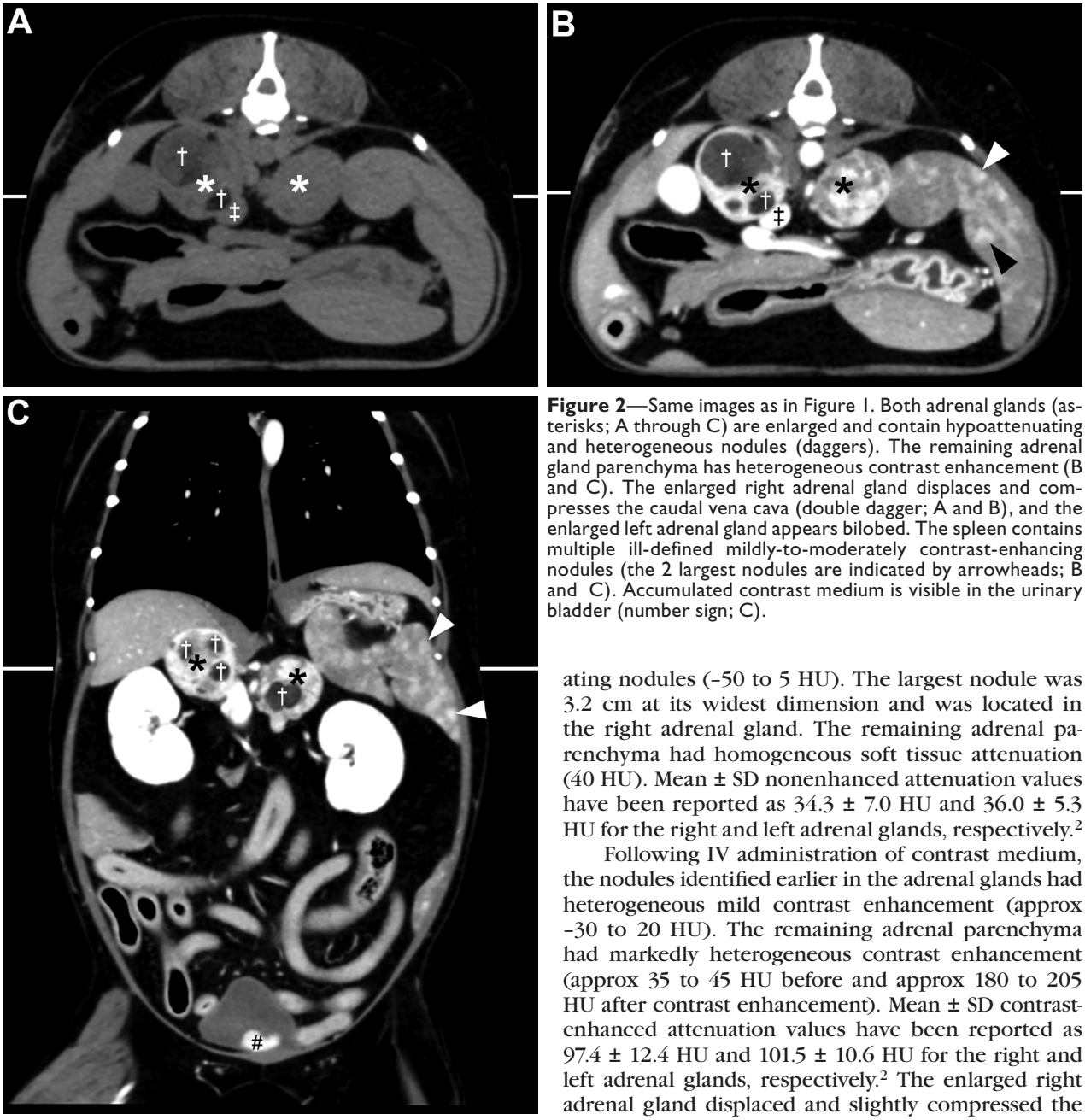

Figure 2—Same images as in Figure 1. Both adrenal glands (asterisks; A through C) are enlarged and contain hypoattenuating and heterogeneous nodules (daggers). The remaining adrenal gland parenchyma has heterogeneous contrast enhancement (B and C). The enlarged right adrenal gland displaces and compresses the caudal vena cava (double dagger; A and B), and the enlarged left adrenal gland appears bilobed. The spleen contains multiple ill-defined mildly-to-moderately contrast-enhancing nodules (the 2 largest nodules are indicated by arrowheads; B and C). Accumulated contrast medium is visible in the urinary bladder (number sign; C).

Diagnostic Imaging Findings and Interpretation

On CT, the right and left adrenal glands were asymmetrically enlarged and had mildly irregular contours **(Figure 2)**. The right adrenal gland appeared round, with a diameter of 3.3 cm (ultrasonographic upper reference limit, 0.62 cm for the caudal pole maximum diameter perpendicular to the longitudinal axis in healthy dogs ≤ 12 kg[1] [26.4 lb]). The left adrenal gland appeared bilobed, with a cranial pole that was 2.6 cm long × 3.0 cm in diameter and a caudal pole that was 0.73 cm long × 0.68 cm in diameter. Both adrenal glands contained multiple round, irregularly marginated, heterogeneous, hypoattenuating nodules (−50 to 5 HU). The largest nodule was 3.2 cm at its widest dimension and was located in the right adrenal gland. The remaining adrenal parenchyma had homogeneous soft tissue attenuation (40 HU). Mean ± SD nonenhanced attenuation values have been reported as 34.3 ± 7.0 HU and 36.0 ± 5.3 HU for the right and left adrenal glands, respectively.[2]

Following IV administration of contrast medium, the nodules identified earlier in the adrenal glands had heterogeneous mild contrast enhancement (approx −30 to 20 HU). The remaining adrenal parenchyma had markedly heterogeneous contrast enhancement (approx 35 to 45 HU before and approx 180 to 205 HU after contrast enhancement). Mean ± SD contrast-enhanced attenuation values have been reported as 97.4 ± 12.4 HU and 101.5 ± 10.6 HU for the right and left adrenal glands, respectively.[2] The enlarged right adrenal gland displaced and slightly compressed the caudal vena cava. There was mild irregular margination of the intraluminal filling with contrast medium in the caudal vena cava adjacent to the right adrenal gland during the venous and delayed venous postcontrast phases. Because there was homogeneous contrast enhancement of the caudal vena cava at that level, findings suggested potential early vascular invasion. In all images of the postcontrast phases, the spleen had multiple mildly-to-moderately contrast-enhancing nodules of various sizes that also had ill-defined margins not visible on the precontrast images. The gallbladder contained a small amount of soft tissue–attenuating, noncontrast-enhancing, dependent material, consistent with sediment. Similar, noncontrast-enhancing, soft tissue–attenuating material was adhered to the gallbladder wall. There was no evidence of obstruc-

tion of the biliary tree, and findings for the remaining abdominal organs, including the liver, were unremarkable. Further, CT of the dog's head (not presented) revealed no abnormalities of the brain or pituitary gland.

On the basis of the various findings, the hyperechoic and hypoattenuating nodules in the adrenal glands were considered most consistent with benign myelolipomas. Given the mass-like appearance and suspected vascular invasion of the caudal vena cava from the right adrenal gland, a concurrent neoplastic process, such as a carcinoma or pheochromocytoma, was considered likely. The abnormal appearance of the left adrenal gland could have been from metastatic disease or a bilateral primary neoplastic process; however, bilateral adenomas and hyperplasia were considered less likely. The splenic nodules were most consistent with a benign process, such as lymphoid hyperplasia or extramedullary hematopoiesis, and less likely from metastatic disease or diffuse neoplastic infiltration. The abnormalities identified in the gallbladder were consistent with an immature mucocele or cholecystitis, alone or in combination.

Treatment and Outcome

Given the bilateral adrenal gland involvement, the suspicion of vascular invasion, and the associated high risk of severe hemorrhage during surgery, medical management was recommended. Treatment with trilostane (1.4 mg/kg [0.64 mg/lb], PO, q 12 h) was initiated.

The owner requested a second opinion at another institution, and several weeks later, surgical removal of both adrenal glands was performed at that institution. Bilateral adrenal excision was successful, and there was no evidence of vascular invasion. Treatment with trilostane was discontinued, and results of histologic evaluation of the excised adrenal glands were consistent with bilateral myelolipomas of the adrenal glands, a benign adrenocortical adenoma of the left adrenal gland, and diffuse adrenocortical hyperplasia of the right adrenal gland. The dog's subsequent hypoadrenocorticism was successfully managed with administration of a combination of prednisone (0.1 mg/kg [0.05 mg/lb], PO, q 12 h) and desoxycorticosterone (1.8 mg/kg [0.8 mg/lb], SC, q 4 wk).

Comments

Adrenal myelolipomas are uncommon, benign, nonfunctional tumors that, to our knowledge, have only been reported in 2 dogs.[3,4] Myelolipomas consist of well-differentiated adipose tissue and normal hematopoietic cells of lymphoid and myeloid lineages. In addition, mineralization is occasionally found in myelolipomas.[5]

The pathogenesis of adrenal myelolipomas remains unclear. In human medicine, adrenal myelolipomas are ultrasonographically well-demarcated, hyperechoic, and heterogeneous masses.[6] In veterinary medicine, splenic myelolipomas are common and appear ultrasonographically as hyperechoic solid nodules.[7] This ultrasonographic characteristic was consistent with findings for the adrenal myelolipomas of the dog in the present report and with findings for another dog with adrenal myelolipomas, from a report[3] that contains, to our knowledge, the only previously described ultrasonographic characteristics of adrenal myelolipomas in dogs. On CT, adrenal myelolipomas appear as well-circumscribed, round or elliptical, hypoattenuating, and heterogeneous nodules or masses in people[6] and dogs,[3] similar to the appearance of the nodules in the dog of the present report. The attenuation values of myelolipomas vary with the proportion of adipose tissue (low attenuation [eg, –120 to –90 HU]) and soft tissue (moderate attenuation [eg, 20 to 40 HU]) present. Therefore, differentiation between an adrenal myelolipoma and a lipid-rich adrenocortical adenoma can be challenging in human medicine. Even if rare, the presence of acute hemorrhage and mineralization (higher attenuation [eg, 60 to 90 and > 100, respectively]) can further complicate differentiation.[6] In dogs, however, adrenocortical adenomas usually do not contain a high amount of fat; nonetheless, the amount of lipid contained in the cytoplasm of adrenal cortical cells can increase with adrenal cortex degeneration, nodular hyperplasia, or cortical adenoma.[5]

Adrenal myelolipomas were identified in combination with adrenocortical adenomas and diffuse adrenocortical hyperplasia in the dog of the present report and in a dog of a previous report.[3] Although rare, a similar combination of abnormalities has also been described in humans.[6] To our knowledge, there have only been 2 previous reports[3,4] that describe adrenal myelolipomas in dogs as bilateral, as in the dog of the present report. In humans, the prevalence of bilateral adrenal myelolipomas is reported as 12% (54/440).[6]

Adrenal myelolipomas, adrenocortical adenomas, and adrenocortical hyperplasia, alone or in combination, should be considered as differential diagnoses in dogs for which CT findings include 1 or more fat-attenuating nodules in either or both adrenal glands.

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