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What Is Your Diagnosis?

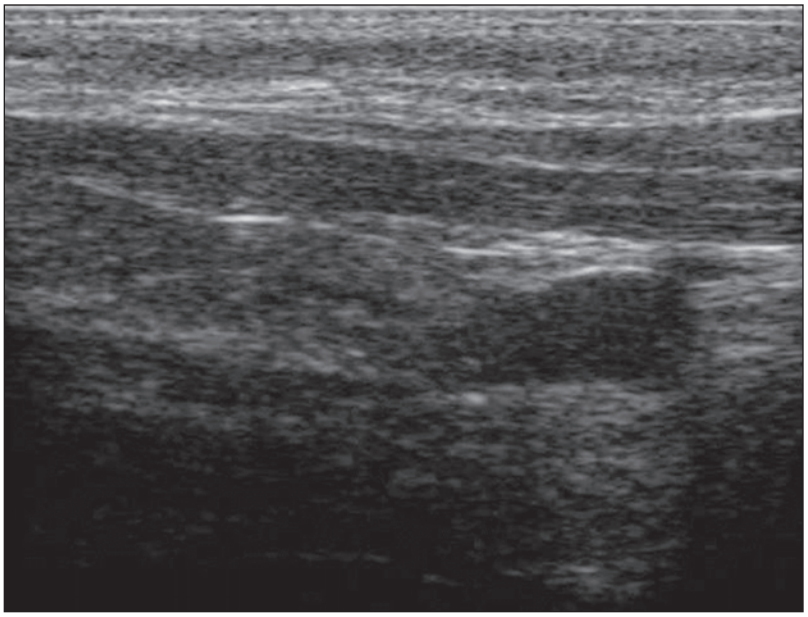

Figure 1—Sagittal ultrasonographic image of the thyroid gland of a 7-year-old neutered male Keeshond evaluated because of listlessness, inappetence, and intermittent vomiting of 6 months' duration.

History

A 7-year-old neutered male Keeshond was evaluated because of progressive listlessness, inappetence, and intermittent vomiting of 6 months' duration. The owners reported dysorexia, lethargy, and muscle weakness. The dog had recurrent cystic calculi (calcium phosphate uroliths) 2 years earlier, and a cystotomy and a urethrostomy had been performed 10 months earlier by the referring veterinarian.

On physical examination, the dog was underweight, and slight bradycardia (92 beats/min; reference range, 100 to 160 beats/min) was detected during auscultation of the thorax. Moderate tartar and gingivitis were also detected. Results of a CBC and serum biochemical analyses indicated mild leukopenia (5,200 WBCs/µL; reference range, 6,000 to 17,000 WBCs/µL), high alkaline phosphatase activity (682 U/L; reference range, 0 to 90 U/L), high calcium (14.4 mg/dL; reference range, 9.0 to 11.9 mg/dL) and parathormone (20.8 pmol/L; reference range, 1.0 to 8.0 pmol/L) concentrations, and low phosphorus (2.48 mg/dL; reference range, 3.0 to 4.7 mg/dL) concentration. Results of urinalysis indicated mild proteinuria (1+ on dipstick) and a low urine specific gravity of 1.017 (reference range, 1.020 to 1.050). Ultrasonography of the thyroid and parathyroid glands was performed with an 8- to 16-MHz linear array transducer **(Figure 1)**.

Determine whether additional diagnostics are required, or make your diagnosis from Figure 1—then turn the page ➧

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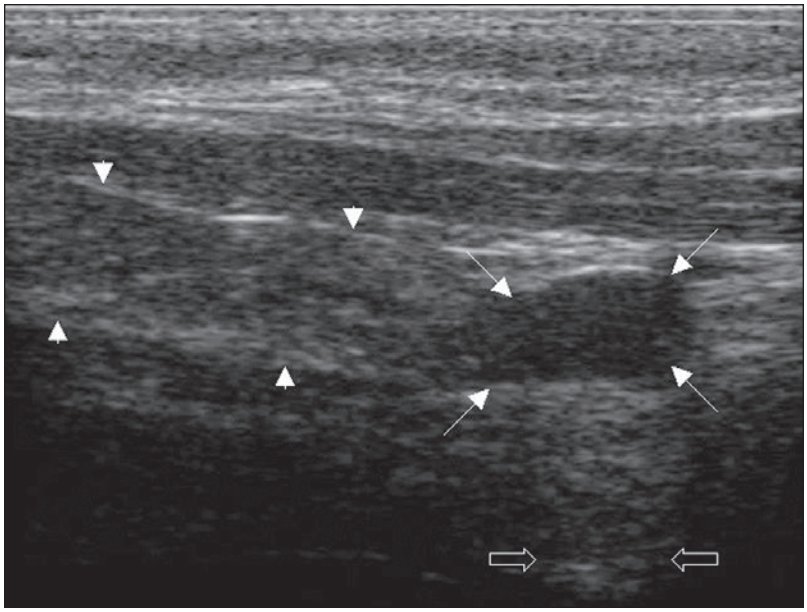

Figure 2—Same ultrasonographic image as in Figure 1. Notice the well-defined, nearly anechoic structure (fine arrows) in the caudal pole of the left thyroid gland (arrowheads) consistent with a large parathyroid gland. Far-field enhancement attributable to the uniformity of the parenchyma of the mass is also evident (open arrows).

Diagnostic Imaging Findings and Interpretation

A well-defined, oval, nearly anechoic structure (6 mm in diameter) is evident in the caudal pole of the left thyroid gland (**Figure 2**). Far-field enhancement attributable to the uniformity of the parenchyma of the mass is also evident. Ultrasonographic findings are consistent with a large parathyroid gland.

Comments

Considering the breed of the dog, results of the serum biochemical analyses, and ultrasonographic findings, the primary differential diagnosis was a solitary parathyroid mass causing primary hyperparathyroidism.

Surgical exploration of the thyroid region was performed. A well-differentiated nodule was identified on the ventral surface of the caudal pole of the left thyroid gland, which was removed via blunt dissection and submitted for histologic examination.

The dog recovered from anesthesia without complications and was treated with fluids IV, cephalexin (20 mg/kg [9.0 mg/lb], IV, q 8 h for 5 days), and morphine sulfate (0.1 mg/kg [0.045 mg/lb], IM, q 6 h for 2 days) after surgery. Serum calcium concentration was monitored every 8 hours for 5 days to prevent hypocalcemia caused by hypercalcemia-induced atrophy of the remaining parathyroid gland. The dog was discharged 7 days after surgery. Results of histologic examination of excised tissue were consistent with an adenoma of the left parathyroid gland.

Primary hyperparathyroidism (PH) can be defined as an excessive and autonomous production of parathormone resulting in chronic hypercalcemia. In dogs, PH is usually caused by a solitary parathyroid adenoma; however, malignant tumors and hyperplasia may develop less frequently.[1,2] Primary hyperparathyroidism is frequently diagnosed in older dogs (7 to 13 years old).[3] Keeshonds appear to be particularly susceptible to parathyroid adenomas.[1]

Unlike other causes of hypercalcemia, PH is usually associated with long-term history of mild or minimal clinical signs including polyuria and polydipsia, inappetence, dysorexia, and muscle weakness, which may be related to persistent hypercalcemia. Recurrent urinary tract infection or calcium-containing urolithiasis may be the only consistent abnormalities.[2,3] The most common clinicopathologic abnormality of PH is persistent hypercalcemia with high serum parathormone concentration and consequently hypophosphatemia; high serum alkaline phosphatase activity is also common.[3]

In dogs, the possible causes of hypercalcemia are numerous; therefore, ultrasonography of the parathyroid glands can be used to differentiate causes of hypercalcemia. Normal parathyroid glands measure ≤ 2 mm and are not routinely visualized during ultrasonographic examination of the cervical region. Large parathyroid glands are usually round or oval and hypoechoic, compared with the surrounding parenchyma of the thyroid gland.[4] Ultrasonographically, parathyroid hyperplasia, adenomas, and adenocarcinomas appear similar. However, the size of the lesion may be used to accurately differentiate neoplastic disorders of the parathyroid gland from hyperplasia. Adenomas and adenocarcinomas are more likely when a solitary parathyroid lesion ≥ 4 mm in diameter is detected, whereas dogs with several lesions ≤ 4 mm in diameter have a high likelihood of having primary or secondary (nutritional or renal) parathyroid hyperplasia.[4]

When no parathyroid gland can be visualized ultrasonographically in a dog with hypercalcemia, hypercalcemia of malignancy should be evaluated by measuring the concentration of parathormone-related protein. Secretion of parathormone-related protein from tumors causes atrophy of the parathyroid glands because of persistent hypercalcemia.

Ultrasonography of the thyroid and parathyroid glands appears to be a valuable tool in differentiating PH, for which surgical resection is generally curative, from secondary hyperparathyroidism or hypercalcemia of malignancy, for which conservative treatment is recommended.

1. Feldman EC. Disorders of the parathyroid glands. In: Ettinger SJ, Feldman EC, eds. *Textbook of veterinary internal medicine.* 4th ed. Philadelphia: WB Saunders Co, 1995;1437–1465.

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3. Berger B, Feldman EC. Primary hyperparathyroidism in dogs: 21 cases (1976–1986). *J Am Vet Med Assoc* 1987;191:350–356.

4. Wisner ER, Penninck D, Biller DS, et al. High-resolution parathyroid sonography. *Vet Radiol Ultrasound* 1997;38:462–466.