

Polyuria & Polydipsia in a Dog

Benjamin Nolan, DVM, PhD, DACVIM (Small Animal)

Aspen Meadow Veterinary Specialists
Boulder, Colorado

Mary Anna Labato, DVM, DACVIM (Small Animal)

Tufts University



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On examination, the patient showed marked truncal alopecia (A) without any evidence of superficial pyoderma (B).

A 10-year-old, 6.8-kg spayed bichon frise was presented for a 2-month history of progressive polyuria and polydipsia and 5-month history of progressive alopecia with intermittent superficial pyoderma.

History

The owner reported that the dog had no vomiting or diarrhea, but its appetite had increased. At routine evaluation 6 months before presentation, the dog weighed 6.3 kg. It was not receiving any medication.

Physical Examination

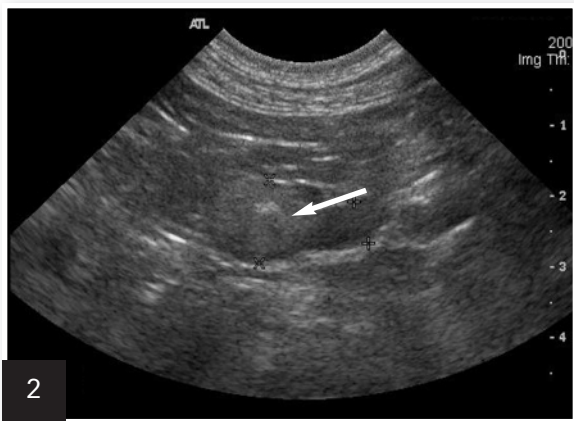
The dog was bright and alert. Vital signs were within normal limits. Physical examination revealed marked truncal alopecia but no evidence of superficial pyoderma (Figure 1). The abdomen appeared rounded; mild cranial organomegaly was present.

Diagnostics & Imaging

Laboratory studies were conducted and evaluated (Table). Adrenocorticotropic hormone (ACTH) stimulation testing showed a baseline cortisol concentration of 4.5 µg/dL (range, 2–6 µg/dL) and post-ACTH cortisol concentration of 9.6 µg/dL (range, 6–18 µg/dL). Low-dose dexamethasone suppression testing revealed a baseline cortisol concentration of 4.5 µg/dL, 4-hour cortisol concentration of 4.3 µg/dL, and 8-hour cortisol concentration of 4.3 µg/dL (range, <1 µg/dL = normal at 4 and 8 hr; 1–1.4 µg/dL = inconclusive; ≥1.5 µg/dL = positive).

Abdominal ultrasonography showed enlargement of the left adrenal gland with a 1.19-cm nodule on the cranial pole (Figure 2). The right adrenal gland appeared normal. The liver was enlarged and hyperechoic.

ACTH = adrenocorticotropic hormone



Abdominal ultrasound showing enlargement of the left adrenal gland with a 1.19-cm nodule (arrow) on the cranial pole

Table Laboratory Results

Parameter	Result (range)
CBC	
Platelets (/μL)	650,000 (140,000–540,000)
Chemistry panel	
ALP (U/L)	2200 (13–289)
ALT (U/L)	197 (14–151)
Cholesterol (mg/dL)	640 (98–300)
Urinalysis (via cystocentesis)	
Specific gravity	1.006
pH	7.0
Protein (mg/dL)	100
WBC (/hpf)	0–2
RBC (/hpf)	0–2

ALP = alkaline phosphatase, ALT = alanine aminotransferase



Ask Yourself

1. What was the most likely diagnosis, and what additional testing should be performed to determine the cause?
2. What caused divergent results on the ACTH stimulation and low-dose dexamethasone suppression tests?
3. What additional diagnostics should be considered to obtain a complete clinical picture?

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Diagnosis

Hyperadrenocorticism

Although the ACTH stimulation test was within the reference range, results of the low-dose dexamethasone suppression test were consistent with a diagnosis of hyperadrenocorticism (HAC). This is particularly notable considering the other supportive findings of polyuria and/or polydipsia, polyphagia,

dermatologic signs, increases in ALP and ALT activities, hypercholesterolemia, thrombocytosis, and hyposthenuria.

Treatment Options

The definitive treatment for an adrenal tumor is surgical removal; however, adrenalectomy is not a straightforward procedure and has a significant mortality rate. Approximately 50% of adrenocortical tumors are benign adenomas; the other half

Did You Answer?



1. The nodule on the left adrenal gland suggested that a functional adrenocortical tumor was the underlying cause of HAC, but additional investigation is required to rule out the more common pituitary-dependent hyperadrenocorticism (PDH).

Approximately 80%–85% of dogs with HAC have PDH; only 15%–20% have an adrenocortical tumor.⁴ In some dogs with PDH, adrenal hyperplasia caused by excessive ACTH stimulation can appear as a nodule. Measurement of endogenous ACTH concentration and a high-dose dexamethasone suppression test can help differentiate PDH from adrenocortical tumor.

This dog had an endogenous ACTH concentration of <10 pg/mL, consistent with a functional adrenocortical tumor as the source of HAC.

2. The divergent results on ACTH stimulation and low-dose dexamethasone suppression testing can be understood after the discovery of the functional adrenocortical tumor. The reported sensitivity of the ACTH stimulation test for detecting this HAC is 61%, indicating that false-negative results are common.⁵ The neoplastic cells in adrenal tumors are abnormal, and although they produce excessive cortisol, they seldom do so in response to ACTH. Therefore, it is not surprising to find a negative ACTH stimulation test with this type of HAC.

The exact sensitivity of the low-dose dexamethasone suppression test for diagnosing adrenocortical tumor has not been reported, but the sensitivity of this test for all types of HAC is 95%. If an ACTH stimulation test is conducted in the diagnostic investigation for HAC, a negative result should be interpreted with caution.

3. Dogs with HAC can have clinically significant comorbid conditions resulting from the effects of increased steroid hormone concentrations. Additional diagnostic testing is recommended.

One study found that 46% of dogs had a urinary tract infection at HAC diagnosis, so a urine culture was indicated.⁶ Of these 46%, fewer than 5% exhibited signs of an infection (eg, pollakiuria, stranguria), and the expected increased WBC counts and bacteria in the urine may not have been present because of the effects of cortisol and urine dilution.

A UP:C should be obtained, but only after the urine culture result is negative to rule out infection as the cause of proteinuria. Hypertension has also been associated with HAC; 86% of dogs have had increased blood pressure at diagnosis.⁷ To document hypertension, blood pressure should be measured on at least two occasions.

This dog had a negative urine culture and a UP:C of 2.8. Its systolic blood pressure measured by Doppler ultrasonography was 180 mm Hg.

Another test to be considered is thromboelastography (TEG). Relatively new in veterinary medicine, TEG has limited availability but is gaining acceptance as a method to evaluate hypercoagulability.

Canine HAC has been associated with thromboembolic complications (eg, pulmonary thromboembolism) that can be severe, and TEG for the detection and treatment of this problem is an active area of research. One study found that 80% of a small population of dogs with HAC had hypercoagulability based on TEG.⁸ However, the precise role of TEG in treatment is still evolving.

A TEG was conducted in this case, and the results were normal. Hypercoagulability was not present on TEG; if it were, treatment with anticoagulants could be considered, but the decision is currently subjective and made on a case-by-case basis.

are malignant carcinomas. Before pursuing surgery, potential metastatic disease should be investigated via thoracic radiography and abdominal ultrasonography. CT scan of the abdomen can also aid surgical planning.

Ideally, HAC is first controlled medically to minimize chance of surgical complications. Trilostane (Veteryl, dechra-us.com) and mitotane (Lysodren) are two commonly prescribed medications. Ketoconazole (Nizoral) is another option.

Dosing schemes for PDH may not effectively treat a functional adrenal tumor. If dose escalation is required, it should be executed cautiously with close monitoring. If surgery is not pursued, long-term medical therapy with these medications is a good alternative. Favorable results have been reported with mitotane or trilostane.¹⁻³

Before surgery was pursued, this dog was treated with trilostane at 1 mg/kg PO q12h. Benazepril was also administered at 0.25 mg/kg PO q12h to address proteinuria and hypertension. After 2 weeks, ACTH stimulation testing showed insufficient control,

and clinical signs persisted. The dose of trilostane was increased by 25% to 1.25 mg/kg PO q12h.

Two weeks later, clinical signs were markedly improved, ACTH stimulation testing showed adequate control of HAC, and systolic blood pressure had decreased to 140 mm Hg. Left adrenalectomy was performed. Recovery was uneventful. Histopathologic examination of the adrenal nodule revealed a benign adrenocortical adenoma.

Follow-up testing (ie, chemistry panel, CBC, ACTH stimulation test) showed that the hyperadrenocorticism and associated signs had resolved. Benazepril was discontinued.

After 1 month, the systolic blood pressure measured by Doppler ultrasonography was 110 mm Hg and urine protein:creatinine (UP:C) ratio was 0.4. ■ **cb**

See **Aids & Resources**, back page, for references & suggested reading.

ACTH = adrenocorticotrophic hormone, ALP = alkaline phosphatase, ALT = alanine transaminase, HAC = hyperadrenocorticism, PDH = pituitary-dependent hyperadrenocorticism, TEG = thromboelastography, UP:C = urine protein:creatinine

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John E. Bauer,
D.V.M., Ph.D.,
Diplomate ACVN

Questions regarding the benefits of fish oil omega-3 fatty acids in canine skin and joint health are some of the most frequent inquiries among pet owners. The following summaries are provided, given the evidence to date.

What are the differences between flaxseed oil and fish oil omega-3 fats for canine skin and hair coat?

The primary fatty acids in flaxseed oil are linoleic (omega-6) and alpha-linolenic (omega-3). While both are polyunsaturated, linoleic acid is regarded as the important skin fatty acid because it contributes an epidermal water barrier function. Studies have shown that while increased amounts of total dietary fat *per se* contribute overall to canine skin health, the polyunsaturated fats provide additional improvements.

Adding alpha-linolenic acid indirectly contributes to the skin water barrier because it results in increased plasma linoleic acid content. Thus, in the case of a dry, dull hair coat without inflammation, adding vegetable oils containing both omega-6 and omega-3 fats is recommended.¹

However, when inflammation is present, such as with atopy or other dermatitis, long-chain omega-3 fatty acids from fish oil (i.e. EPA and DHA) are needed. These omega-3s provide an anti-inflammatory effect. They also help to protect the skin from day-to-day scrapes and scratches. In this way, feeding dietary omega-3 fatty acids helps balance inflammation and assures healthy canine skin and hair coat beyond vegetable oil sources.²

How do fish oil omega-3 fatty acids benefit joint health and osteoarthritic conditions?

Chronic, progressive conditions such as osteoarthritis are frequently characterized by the production of cytokines and other inflammatory mediators that contribute to advanced joint capsule tissue destruction and pathology. Fish oil omega-3 fatty acids are well known for their ability to provide anti-inflammatory precursors at the cellular level to help mitigate these processes. To do so, however, fish oil omega-3s must be present daily and in high enough dosages to provide such an effect.²

References:

1. Kirby NA *et al.* (2009) *J An Physiol An Nutr* 93, 505-511.
2. Heinemann KM *et al.* (2005) *J Nutr* 135, 1960-1966.