

Pacific Tide

An informational newsletter

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About our Author

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Dr. Garcia started his undergrad education at the University of Georgia and finished his undergrad work at UCLA, after moving to California in 2001. He received his Doctorate in Veterinary Medicine at UC Davis in 2008. He then did a year-long rotating internship at The University of Minnesota in St. Paul, MN before returning in 2009 to UC Davis for an internal medicine residency. He became board certified in internal medicine in 2012. Dr. Garcia has special interests in gastroenterology, urology endoscopy and fungal diseases. He shares his life with his wife Catherine, two children, a dog, cat, and a parrot. In his spare time he enjoys backpacking and taking his children to explore tide pools.



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Addison's Disease

Hypoadrenocorticism (Addison's disease) is an uncommon endocrine disease of dogs. Hypoadrenocorticism is due to a lack of hormone production by the cortex of the adrenal glands. Immune destruction of the adrenal cortex is by far the most common cause of hypoadrenocorticism, though lack of ACTH production by the pituitary (secondary Addison's) or other causes of adrenal destruction have been reported.

The two hormones that are produced by the adrenals that are involved in Addison's disease are cortisol and its relatives (so called glucocorticoids, because they induce glycolysis and gluconeogenesis) and aldosterone (a type of mineralocorticoid). Glucocorticoids are involved in numerous processes in the body, though most of the noted clinical effects of deficiency are gastrointestinal, as cortisol is required for appropriate GI function and enterocyte health. Aldosterone's main action is to cause sodium (and passively, chloride) retention and potassium excretion by the kidneys. Aldosterone also helps maintain blood pressure and water balance through direct and indirect effects.

Classic, or typical, hypoadrenocorticism is manifested by deficiencies in both glucocorticoids and mineralocorticoids. Females are over represented (~70% of cases) and it is usually a disease of younger to middle aged dogs (75% are <7 years of age). Overrepresented breeds include Poodles, Great Danes, Westies, Portuguese Water Dogs, Nova Scotia Duck Tolling Retrievers and Bearded Collies. Dogs with typical Addison's may present with vague signs of lethargy, inappetence and accompanying diarrhea/vomiting (often bloody), or they may present collapsed and in hypovolemic shock. Polyuria/polydipsia may also be reported. The signs displayed are variable as the degree of hypovolemia is mainly due to the electrolyte disturbances from a lack of aldosterone (which is often exacerbated by vomiting and diarrhea due to cortisol deficiency).

Lab work abnormalities in typical Addisonian patients is quite variable, and changes may be marked or minimal. CBC often lacks the neutrophilia and lymphopenia of an expected stress leukogram, and uncommonly, an inappropriate lymphocytosis or eosinophilia is seen. The hallmark, classic lab finding in a typical Addisonian patient is a hyponatremia and hyperkalemia caused by a lack of aldosterone. These changes are often marked. Some advocate using the Na:K ratio to as a screen for hypoadrenocorticism, where Na:K <27 is suspicious for Addison's, and <23 highly consistent. Other changes on chemistry variably seen include: azotemia (often severe), hyperphosphatemia, hyper/hypocalcemia, hypoalbuminemia, hypocholesterolemia, and hypoglycemia. Urine generally reveals a specific gravity of <1.030 (often isosthenuric at 1.008-1.012), often causing a misdiagnosis of acute kidney injury despite this azotemia being almost exclusively volume responsive (prerenal).

Diagnosis of typical Addison's requires an ACTH stim(ulation) test with cortisol measurement to confirm a lack of adrenal response. A resting cortisol level (discussed below) can be run as a screen prior to ACTH stim, but this step is often skipped with classic lab changes and a critical patient. Treatment will not be discussed in detail here, but involves aggressive IV fluid therapy and acute and chronic supplement-

tation of glucocorticoids (usually dexamethasone in-hospital at 0.15 mg/kg/day various routes) and mineralocorticoids (DOCP 2.2 mg/kg IM/SQ q25+ days or fludrocortisone 0.01-0.02 mg/kg/day PO).

Atypical hypoadrenocorticism generally involves only a deficiency in glucocorticoids, but aldosterone production is maintained. It is rare, and only accounted for 5% (10/225) of all cases of hypoadrenocorticism in one study. Even more rare is mineralocorticoid deficiency with glucocorticoid production preserved (2 cases in the veterinary literature).

Atypical Addison's is usually due to selective destruction of the cortisol-producing part of the adrenal glands or rarely, secondary hypoadrenocorticism. Atypical hypoadrenocorticism has many similarities to classic hypoadrenocorticism, though the disease is often more insidious and has a longer time to diagnosis. Similar breed predisposition has been recognized for atypical as classic Addison's, and females seem equally over represented. Studies vary as to the average age of diagnosis of atypical Addison's. An earlier study found atypical dogs were older (7 years vs 4.6 years of age for typical), though a more-recent study found a median age of only 3.6 years. Clinical signs of atypical Addison's tend to be vague, but generally involve chronic, waxing and waning GI manifestations (inappetence, vomiting, diarrhea) and nonspecific lethargy. Hypovolemic shock is not generally seen, as aldosterone is present to prevent sodium wasting and circulatory collapse. Because of these milder, nonspecific signs, atypical Addison's has a longer time to diagnosis (4.4 months vs 1.2 for typical).

CBC findings with classic and atypical Addison's are often similar, with a cortisol-driven stress leukogram generally absent. Chemistry finds essentially normal electrolytes (Na:K > 27, median was 38). Hypocholesterolemia is more common with atypical Addison's, and many of the other glucocorticoid-dependent findings, such as hypoalbuminemia and hypoglycemia, are often seen. Azotemia and hyperphosphatemia are generally not seen, as hypovolemia is generally not present.

Diagnosis of atypical hypoadrenocorticism is identical to typical, involving an ACTH stim that finds a lack of post ACTH cortisol production. Because of the cost of synthetic ACTH, many clinicians perform a random/basal cortisol as a screen in patients with possible atypical Addison's. A random (i.e. "resting") cortisol level of >2.0 has been shown to essentially rule out hypoadrenocorticism (<1% chance of having Addison's). If the cortisol returns \leq 2.0, an ACTH stim to confirm or rule out hypoadrenocorticism is required. In the exceedingly rare case of aldosterone-deficient Addison's disease, an ACTH stim with pre/post aldosterone levels would be required to show a lack of aldosterone production.

Treatment of atypical Addison's involves only glucocorticoid supplementation (usually prednisone) as aldosterone production is preserved. Prednisone dose varies by the patient, but is usually in the range of 0.1-0.33 mg/kg/day, though it may be lower. This is titrated to the minimally effective dose to avoid relapse of GI signs. The prognosis with treatment is excellent.

It is commonly believed that almost all dogs with atypical Addison's will progress to classic as immune destruction ultimately leads to aldosterone deficiency. This was not supported in one retrospective study, where only one (of 7) became aldosterone deficient. Despite this, checking electrolytes initially monthly following diagnosis is prudent, though this can be decreased to q3-6 month rechecks if the patient is doing well clinically and electrolytes appear stable.

Though uncommon, hypoadrenocorticism should be considered in any dog with chronic GI signs, or acute decompensation with appropriate lab findings. Despite the frequent delay in diagnosing this condition, the prognosis is excellent once made.

Our Doctors

Internal Medicine

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Michelle Pressel, DVM, DACVIM (SAIM)
Ryan Garcia, DVM, DACVIM (SAIM)

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Tom LaHue, DVM, DACVS
Dean Filipowicz, MS, DVM, DACVS

Oncology

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Lillian Good, DVM, DACVECC

Cardiology

Mandi Kleman, DVM, DACVIM(Cardiology)

Dermatology

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Radiology (VRS)

Larry Kerr, DVM, DACVR
Mark Lee, DVM, DACVR

Emergency

Christian Robison, DVM
Kim Delkener, DVM
Mark Saphir, DVM
Jessica Kurek, DVM

Behavior

Jan Brennan, DVM (practice limited to behavior)

About Our Hospitals

Pacific Veterinary Specialists was founded to provide high quality, specialized medical care to companion animal patients. Our practice is dedicated to serving the veterinary community as a partner in total patient care. We offer comprehensive specialized services including endoscopy, Doppler ultrasound, surgery, 24-hour ICU care, and emergency and critical care. Our staff is committed to providing compassionate and thorough medical care that meets the needs of the patient, client, and referring veterinarian. In September 2011 we opened PVSM and offer internal medicine, oncology, dermatology and cardiology Tuesday through Thursday in Monterey. Behavior consultations by appointment are available on Mondays.

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