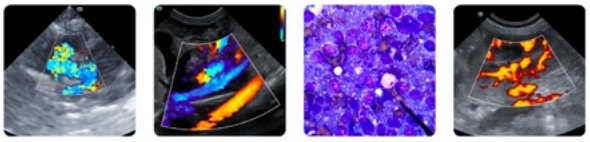

PATIENT
[redacted]

SPECIES
Canine

BREED
Pit Mix

SEX
Spayed female

AGE
8 years

WEIGHT
69.8 lbs

INTERPRETED BY
Eric Lindquist, DMV DABVP, Cert. IVUSS

IMAGING PERFORMED BY
Dr. Wasserman

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REFERRING VET
[redacted]

INVOICE
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DATE
[redacted]

PRESENTING CLINICAL SIGNS

[redacted]The patient has a history of severely chronic polyuria/polydipsia (PU/PD) and was suspected to have urinary incontinence. The patient has been treated with Proin. Last year, ALT was elevated at 400 U/L, with no other concurrent biochemical abnormalities. Follow-up bloodwork normalized while receiving Denamarin therapy. Updated bloodwork and urinalysis were submitted today. Performance of an LDDST and urine metanephrine testing is being considered pending review of the abdominal ultrasound findings and specialist report from SonoPath. Blood pressure to be obtained tomorrow and echocardiogram submission closer to the time of referral if staff elects further workup.

Abnormal PE/Chem/CBC/UA Results:

The patient was sedated for diagnostics with 0.2 mL Dexdomitor (0.5 mg/mL) IV. Thoracic radiographs showed no evidence of metastatic disease. Preliminary echocardiographic evaluation indicated cardiac function within normal limits, and no cardiac murmur was auscultated. The patient has a history of persistently dilute urine without evidence of urinary tract infection, with urine specific gravity ranging from approximately 1.013 to 1.030 on multiple urinalyses. A cystocentesis sample was obtained today for urinalysis and additional testing. Current bloodwork and urinalysis are pending. Rectal examination was within normal limits today.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. The pelvic urethra was imaged 2.0 cm beyond the cystourethral junction and appeared normal. The ureters were not visible which is normal. No uroliths or sediment were visualized and anechoic urine was present. No evidence of inflammatory or neoplastic changes was noted. Ureteral papillae were normal.

The **kidneys** revealed normal size and structure, corticomedullary definition and ratio for this age. The cortices presented largely uniform texture with normal echogenic relationship to liver and spleen. Medullary structure differed distinctly from the cortex. The capsules were acceptably uniform without significant irregularities. The right kidney measured 6.8 cm with slight pyelectasia. The left kidney measured 6.58 cm.

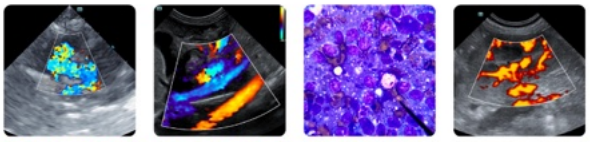
Adrenal Glands

The **left adrenal gland** was visualized and recognized as having normal shape, size, position and echogenicity for this breed. The phrenic vasculature, glandular echogenicity and detail were unremarkable. Capsule, cortex, and medullary definition were normal for this age patient. The left adrenal gland measured 0.74 cm at the caudal pole and 0.53 cm at the cranial pole.

The **right adrenal gland** revealed an expansive, hypoechoic mass that measured 4.5 cm. The mass invaded the phrenic vein and vena cava. CVC invasion was approximately 1.5 cm. The mass was significantly vascular on color Doppler assessment.

Spleen

The **spleen** was largely smooth with subtle heterogeneous parenchymal changes while maintaining normal echogenic relationship to the liver and kidney. These changes are consistent with normal age-



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related alteration. The capsule was smooth without noticeable impingement from within the spleen or from pathology in the adjacent abdomen. The splenic vasculature demonstrated normal volume without signs of congestion or significant contraction. No evidence of active acute or chronic inflammatory, neoplastic, or infarctual changes was noted.

Liver

The **liver** images from right and left intercostal as well as subcostal views revealed subjectively normal liver size, contour, and structure. Some age-related parenchymal remodeling was noted but likely not clinically significant at this time. Vascular and biliary tracts were of normal volume and no evidence of congestion was noted. The gallbladder presented some dependent debris with essentially normal contour. The cystic and common bile ducts were normal. No overt evidence of active inflammatory, infiltrative or regenerative pathology was noted but should be paired with current or past LE elevations regarding any clinical significance to this presentation. The hepatic lymph nodes were unremarkable.

Gastrointestinal

A mild amount of fluid was noted in the **stomach** without evidence of peristalsis. Normal curvilinear patterns were maintained throughout the GI tract. No evidence of foreign body. A minor amount of stasis was noted in the stomach. The small intestine and colon presented with normal curvilinear patterns and no evident pathology. This presentation is most consistent with gastric ileus or idiopathic stasis.

Pancreas

The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

Free Abdomen

An accessory splenic nodule was noted in the left cranial abdomen measuring 2 cm. FNA is recommended to confirm.

ULTRASONOGRAPHIC FINDINGS

Invasive right adrenal mass, potentially still resectable.

Likely concurrent gastritis.

Suspected accessory splenic nodule.

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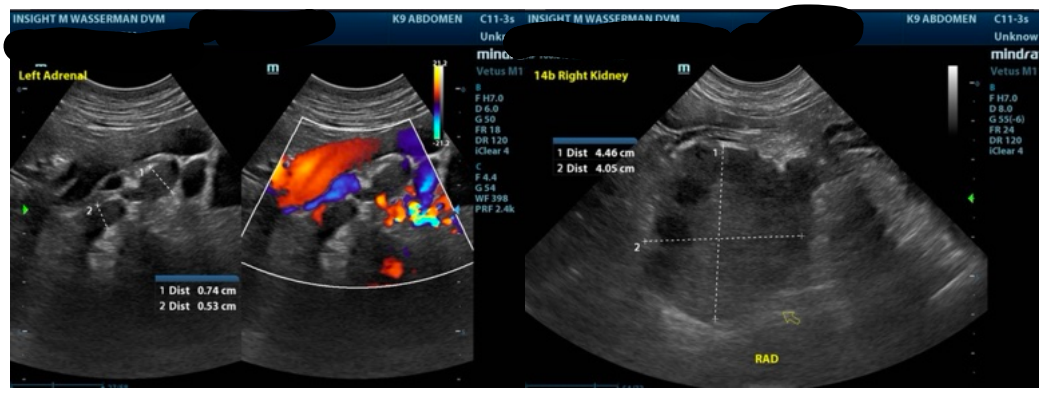
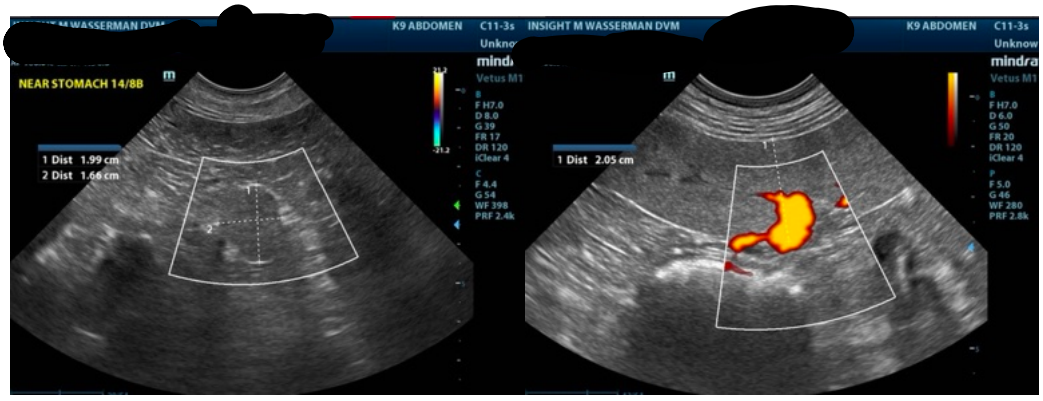
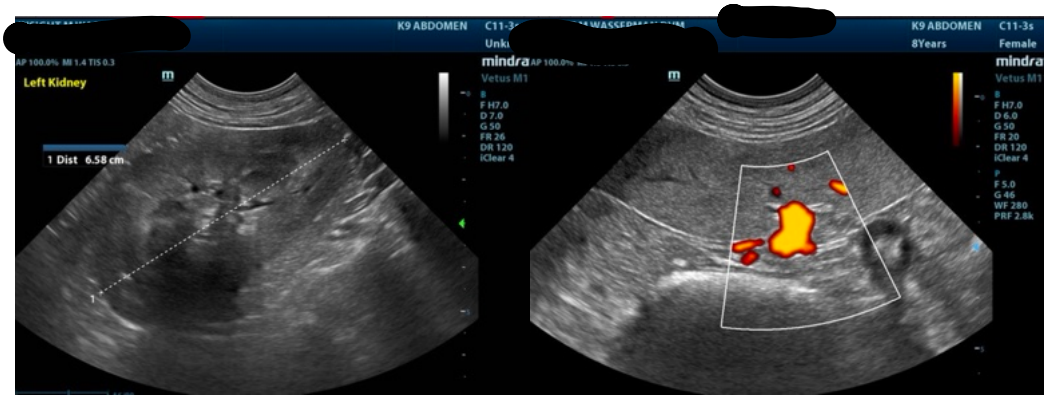
INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Pheochromocytoma is highly suspected, carcinoma is possible. CT evaluation for surgical planning, surgical consult is indicated. FNA of the accessory splenic nodule is recommended for confirmation.

Serial blood pressure measurements are recommended in this patient. If hypertension is an issue metanephrine level is recommended. If the patient appears Cushingoid and urine specific gravity is less than 1.020 then work-up for adrenal dependent Cushing's is indicated. Recheck is recommended in 2-3 weeks to assess for any progression of the adrenal gland.







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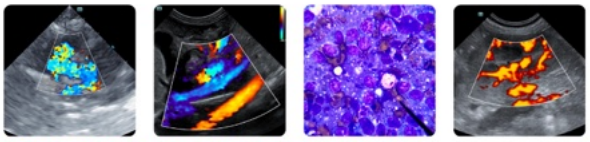

Clinical Sonography & Telecytology
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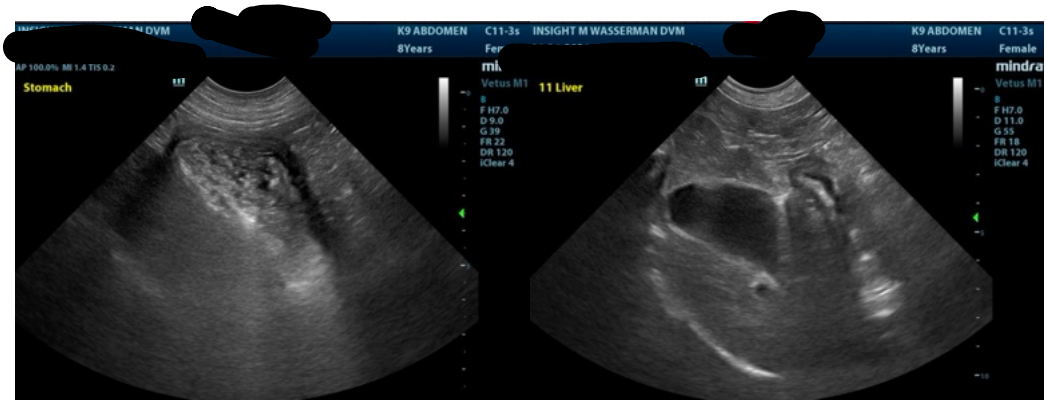

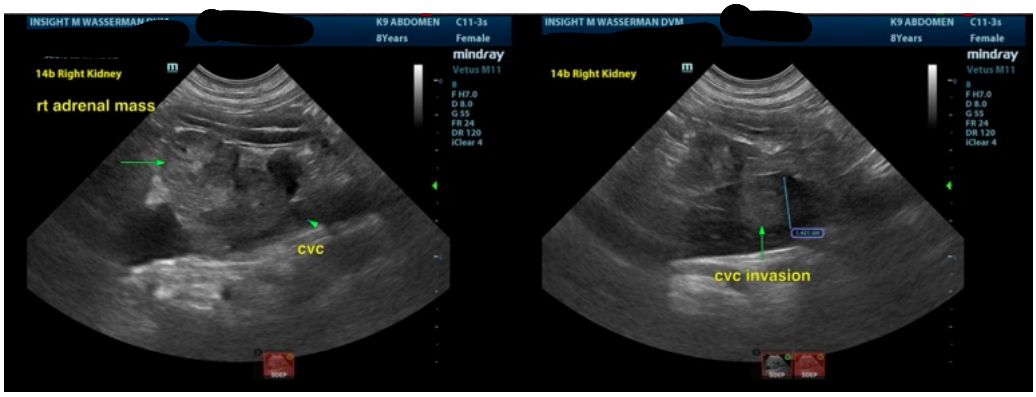

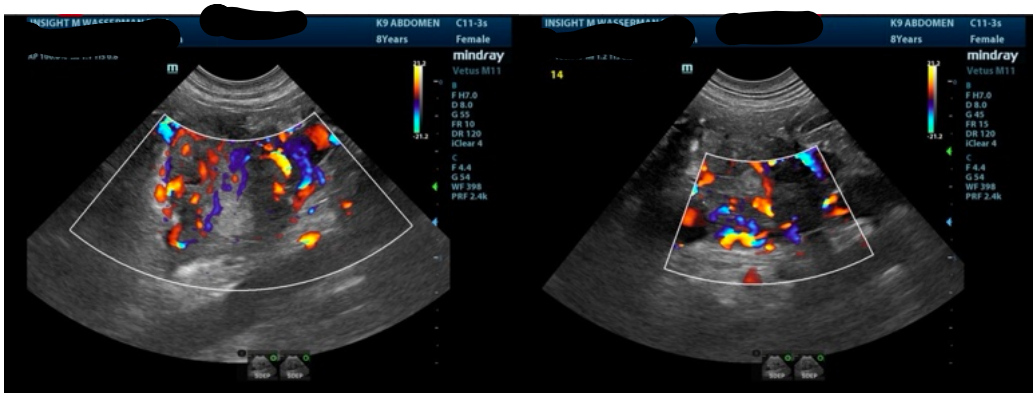

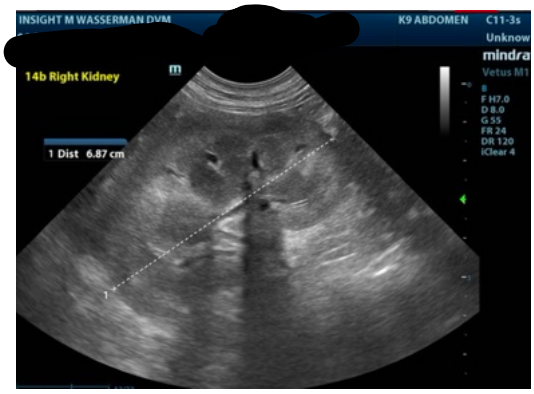

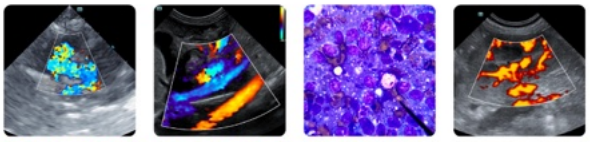

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The information and recommendations provided are based on the images presented by the referring veterinarian/sonographer. No evaluation can be communicated regarding pathology that was not visible in the image/video clips provided.

Thank you for this referral. If the clinical or image interpretation does not parallel your findings or if I can be of any further assistance please contact me.

Eric Lindquist, DMV, DABVP (CFM), Cert. IVUSS, CEO of SonoPath.com

info@SonoPath.com

Excerpt from the Curbside Guide: https://sonopath.com/thecurbsideguide/

Adrenal Tumors

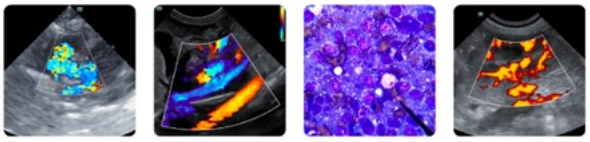
DESCRIPTION An adrenal mass is suspected when the maximum width of the adrenal gland exceeds 1.5 cm, there is loss of normal architecture or shape, or the shape or size between the affected adrenal gland and the contralateral gland is asymmetrical. The latter comprises the initial criteria for diagnosis; however, a bulbous enlargement of the cranial or caudal pole of the adrenal gland is common in dogs with no adrenal pathology and can be misinterpreted as an adrenal mass.

If the suspected mass is not precipitating obvious signs (i.e., aggressive behavior), then an abdominal ultrasound should be repeated to confirm that the mass is a consistent finding before pursuing further diagnostics or surgery. Large breed dogs (Poodles, German Shepherds, Retrievers, and Terriers) and female dogs appear to be overrepresented in the clinical reviews of adrenal tumors. Adrenal tumors in cats are rare with minimal information to characterize the disease. However, adrenal carcinoma and aldosterone-producing tumors are the more common adrenal masses in our archived feline population. More specific information regarding this pathology may be found in the feline hyperaldosterone section of this chapter.

Incidental adrenal lesions should be investigated clinically if discovered on ultrasound. Non-neoplastic adrenal lesions, such as cysts or granulomas, are very rare in dogs and cats, and the high incidence of metastatic lesions justifies a thorough hormonal screening as well as evaluation for nonadrenal neoplasms. Although incidental adrenal masses may appear to be nonfunctional at the time of diagnosis, if fact, it seems more likely that they are subclinically functional. The diagnosis of functional adrenal tumors is discussed below; however, the identification of a nonfunctional, incidental adrenal mass creates a management dilemma.

CLINICAL SIGNS Clinical signs attributable to adrenal tumors are dependent on hormone secretion type. Please see below.

DIAGNOSTICS Cortical adrenal tumors, such as adenomas and adenocarcinomas, are responsible for 15–20% of hyperadrenocortical cases in dogs, specifically adrenal-dependent hyperadrenocortism (ADH). Although ADH cases are usually unilateral, up to 20% can be bilateral. The tumor may invade the aorta on the left or the vena cava on the right and can metastasize to the liver and lungs. Practitioners must differentiate ADH masses from hyperplastic, nonfunctional, benign adrenal tumors, as well as pheochromocytomas. Thus, dynamic function tests (i.e., LDDS, HDDS, ACTH stimulation, ACTH baseline, urine cortisol:creatinine ratio) are essential, as is conducting routine biochemistry (ALP is elevated in more than 90% of cases) and urinalysis (true polyuria/polydipsia [PU/PD] with USG < 1.020) to adequately determine the need for surgical intervention or aggressive medical therapy. It is important to assess the following: blood pressure for hypertension, oscillating hyper- and hypotensive episodes in cases of pheochromocytomas, urine protein:creatinine ratios, and serum antithrombin III to determine the risk for thromboembolism. Moreover, it is essential to evaluate the entire clinical picture and objective probabilities of possessing a true hyperadrenocorticism case. This further entails ruling out other sources of PU/PD, such as primary polydipsia, renal disease, electrolyte abnormalities, infections, and diabetes insipidus or mellitus.



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Canine

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Pit Mix

SEX
Spayed female

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How to decide malignant or benign, functional or nonfunctional In some cases, it may be difficult to determine whether the mass is malignant or benign, functional or nonfunctional, prior to surgical removal and histopathological examination. A thorough review of the clinical signs, physical examination findings, routine blood work, urine tests, and appropriate hormonal tests should be conducted to determine the functional status of an incidental adrenal mass.

Malignancy is more often associated with larger masses. The larger the mass, the more likely metastasis has already occurred, despite a lack of detectable lesions on ultrasound and thoracic radiographs. Invasion of the mass into surrounding organs or blood vessels also supports malignancy, as does the detection of additional mass lesions with abdominal ultrasound and thoracic radiographs. Use of imaging modalities, such as CT and MRI, will likely provide additional data on the characteristics of specific adrenal lesions for use in diagnosis and treatment planning.

Ultrasonography is the primary instrument for assessing tumor size, aggressiveness, non-capsulated versus capsulated appearance, vascular invasion, and hepatic or other metastasis. Ideally, the patient will have fasted prior to the ultrasound; one may choose to administer an enema to enhance visibility around the ascending and descending colon. Ultrasound-guided biopsy or fine-needle aspiration (FNA) may be possible on the larger masses, especially on the left side; however, adjacent vascular structures often prevent the feasibility of this procedure, and it is an operator-dependent maneuver.

Diagnosis of the functional adrenal mass

I. Cortisol-secreting It is very rare that a patient with hyperadrenocorticism will have a repeatable urine specific gravity greater than 1.020, so it must be determined whether the patient is truly PU/PD. If yes, then dynamic function testing is appropriate. If the patient is not truly PU/PD, then a false positive result must be considered before treatment is initiated as the resulting hypoadrenocorticism can be life threatening. Other causes of dysuria, such as occult urinary tract infection, must then be considered. The most common functional adrenal tumor identified in dogs and cats results in hyperadrenocorticism. Approximately 15% of hyperadrenocorticism cases will be caused by a functional adrenal tumor, of which 50% of these will be malignant.

A. Clinical signs can include PU/PD, polyphagia, abdominal distention, bilaterally symmetrical truncal alopecia, delayed fur regrowth, hyperpigmentation, comedones, calcinosis cutis, excessive bruising, poor wound healing, ectopic calcification of kidneys and blood vessel walls, pyodermas, muscle weakness, exercise intolerance, hypertension, and panting.

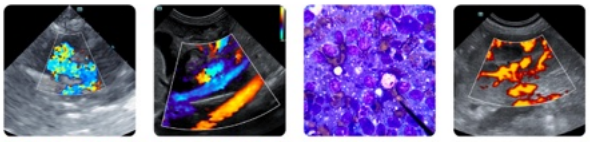
B. Ultrasound usually reveals a small or atrophied contralateral adrenal gland as a result of suppressed pituitary ACTH secretion. Bilateral disease occurs in 10–20% of cases. Adenomas of the adrenal gland are generally less than 2 cm in diameter, and carcinomas can be any size (often they are >2 cm). Calcification does not appear to be predictive for either adenoma or carcinoma.

C. Specific biochemical tests: urine cortisol:creatinine ratio, ACTH stimulation test, and LDDS test.

II. Catecholamine-producing Pheochromocytoma is a tumor derived from the chromaffin cells of the adrenal medulla; it is relatively common in dogs, but it is quite rare in cats. These cases should be considered malignant until proven otherwise. Invasion/entrapment/compression of the caudal vena cava is common. Mural invasion or luminal narrowing of the aorta, renal vessels, adrenal vessels, and hepatic veins may also occur.

A. Clinical signs associated with this type of tumor are usually related to the invasion of local structures, metastases, or the secretion of catecholamines. The most common clinical signs of excess catecholamines include generalized weakness, episodic collapse, tachypnea, panting, tachycardia, and cardiac arrhythmias. Catecholamine release and hypertension tends to be episodic; thus, failure to document systemic hypertension does not rule out pheochromocytoma.

B. On ultrasound, the contralateral adrenal gland is usually normal in size and shape. Pheochromocytomas do not typically calcify readily.



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C. Tests: Many of the clinical signs and blood pressure alterations are similar for pheochromocytoma and ADH. Therefore, it is important to rule out ADH before focusing on pheochromocytoma. The diagnosis prior to surgery is primarily one of exclusion. Measuring urinary or plasma catecholamine concentrations or their metabolites can be used to diagnosis a pheochromocytoma. Urine metanephrines and normetanephrines, which are metabolites of catecholamines, serve as stable markers that can indicate the presence of a pheochromocytoma.

III**. Aldosterone-secreting (rare in dogs and cats)**

A. Clinical signs (Conn's syndrome) are related to excessive secretion of aldosterone which causes sodium retention and potassium depletion. The resulting symptoms include lethargy, weakness, mild hypernatremia, severe hypokalemia (usually < 3.0 mEqu/L), with consequential cervical ventroflexion, and systemic hypertension.

B. Ultrasound usually reveals a normal contralateral adrenal gland.

C. Tests: Documenting increased plasma aldosterone concentrations before and after ACTH administration is a means of confirming the diagnosis. If weakness and severe hypokalemia are present, plasma aldosterone concentrations can be measured along with plasma cortisol concentrations during the ACTH stimulation test.

IV. Progesterone-secreting Although a functional tumor arising from the zona reticularis of the adrenal cortex could secrete excessive amounts of estrogen, progesterone, or testosterone, to date only progesterone-secreting adrenocortical tumors in cats have been documented.

A. Clinical signs include diabetes mellitus and feline fragile skin syndrome, which is characterized by progressively worsening dermal and epidermal atrophy, patchy endocrine alopecia, and easily torn skin.

B. Ultrasound usually reveals a normal contralateral adrenal gland.

C. Tests: Diagnosis requires documenting an increased plasma progesterone concentration. The clinical features mimic feline hyperadrenocorticism, which is the primary differential diagnosis. Pituitary-adrenocortical axis test results are normal-to-suppressed in cats with progesterone-secreting adrenal tumors.

V. Deoxycorticosterone-secreting (rare)

A. Clinical signs are related to mineralocorticoid activity and include weakness, marked hypokalemia, and systemic hypertension.

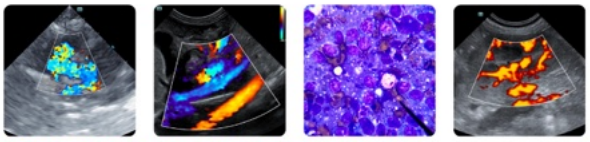
B. Tests: Increased plasma deoxycorticosterone and non-detectable plasma aldosterone concentrations have been documented in dogs.

VI. 17-OH-progesterone-secreting (rare)

A. Clinical signs are similar to hyperadrenocorticism.

B. Pre- and post-ACTH stimulation plasma 17-OH-progesterone concentrations will be increased.

TREATMENT If hormonal tests for ADH and serum electrolytes are normal and clinical signs suggestive of pheochromocytoma are present, one can assume the adrenal mass is a pheochromocytoma and begin treatment with an α-adrenergic antagonist (ex., phenoxybenzamine at 0.25 mg/kg PO BID initially) for at least two weeks to prevent severe clinical manifestations of hypertension and to promote a smooth anesthetic induction if adrenalectomy is planned. Adjustments to the dose are based on clinical response; an increase in the dose should be considered if clinical signs do not improve after two weeks of treatment. If hormonal tests for ADH and serum electrolyte concentrations are normal, clinical signs suggestive of pheochromocytoma are not present, but an adrenalectomy is nevertheless planned, one should still assume the adrenal mass is a pheochromocytoma and begin phenoxybenzamine treatment prior to adrenalectomy.



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When a cortisol-producing adrenal tumor has been documented, medical therapy with trilostane (5–20 mg/kg PO Q24hr) or mitotane (25–50 mg/kg PO Q24hr for 10 days, then every 4–7 days) should be considered.

The biggest dilemma is whether to perform an adrenalectomy if hormonal tests for hyperadrenocorticism and serum electrolyte concentrations are normal, and clinical signs and systemic hypertension suggestive of pheochromocytoma are not present.

An aggressive approach (adrenalectomy) assumes that the mass is malignant until proven otherwise and should be removed before metastasis has occurred. In theory, this approach would offer the best chance for long-term survival; however, the age of the patient, the size of the mass, the presence of concurrent diseases, the level of invasion into other organs, and the probability that metastases already exist should factor into the decision. Poor surgical candidates generally include dogs compromised from the effects of hypercortisolism, older animals, animals with comorbidities, those for whom invasion has been aggressive and surgical or post-surgical complications are likely, animals with very large masses that have likely already metastasized, and those with documented potential metastatic disease. In addition, adrenalectomy may not be indicated when the mass is small (< 3 cm diameter) and nonfunctional and the patient is healthy. Reports suggest that there is an approximate 45% success rate of surgical resection of adrenal masses with a positive prognosis inversely proportionate to tumor size.

In cases of concurrent hepatic nodular changes, liver biopsy samples can be obtained during surgery when suspicious lesions are visualized by ultrasound. Hyperadrenocorticism often causes benign nodular hyperplasia of the liver and should not be automatically interpreted as a sign of hepatic metastasis during ultrasonographic examination. Rather, suspect lesions should be confirmed and biopsied either at surgery or via ultrasound-guided FNA or core biopsy. Post-operative complications include delayed wound healing due to excessive corticoid circulation and wasting, hemorrhage, sepsis, and thromboembolism.

When surgery is a risk and a functional adrenal tumor has been documented, medical therapy as outlined above should be considered. Medical therapy will not impede metastatic events. An alternative approach in these cases is to determine the rate of growth of the mass by repeating abdominal ultrasounds initially at two, four, and six months. If the adrenal mass does not change in size, the time between ultrasound evaluations can be increased to every 4–6 months; however, if the adrenal mass is increasing in size, adrenalectomy should be considered.

CONCLUSION Any incidentally discovered adrenal tumor warrants investigation into functionality and metastasis. The course of treatment for each case depends largely on which hormones are secreted by the adrenal tumor. Each case should be carefully evaluated on an individual basis before adrenalectomy is considered for aggressive tumors.

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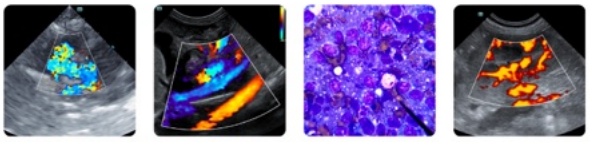

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4. Nelson, RW. Diagnostic approach to the incidental adrenal mass. World Small Animal Veterinary Association World Congress, Granada, Spain, 3-5 October, 2002.

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