Imaging4Pets™
Mobile Ultrasound Veterinary Support

IMAGING REPORT

Patient Information

Patient:		Patient ID:		Birth Date:	2011-04-07
Age:	13Y	Weight (kg)	76.4 Lbs	Modalities:	US
Patient Species:	Canine	Patient Breed:	American Staffordshire Terrier	Gender:	F Altered
Study Description:		Study Date:	2024-06-05	Study Time:	10:15:40
Referring Physician:		Sedation:	No	Anesthesia:	No

Requester

Institution:	Imaging4Pets	Submit Date:	2024-06-05 14:56:31 UTC
Report By:		Billing Code:	AUS

Annotated Images Requested: No

Request Priority: Normal

History

On Fluoxetine 60mg & Cosequin daily. CBC/Chem/T4=wnl. UA= UTI, treated with Clavamox
Reason for Exam
Pot bellied appearance
History and Concurrent Illness
Bursts skin cysts.
Differentials
Normal liver v. early liver disease
Prior Imaging Impressions
Liver borderline/mildly enlarged on radiographs based on my interpretation & supported by SignalPet AI.
Other Comments
Current on rabies, DHPPL, bordetella. Fecal negative & heartworm+3tick test negative.

Findings

Liver: Subjectively enlarged in size (liver size more accurately determined via radiographic study). Mildly heterogenous appearance. No nodules, masses, or cysts present.
Gallbladder: Contains anechoic fluid and has a mild to moderate amount of echogenic debris much of which appears less dependent and potentially organizing/solidifying. GB wall is at high end of normal (0.20 cm). The GB fossa and adjacent tissues are normal.
Spleen: Small hyperechoic nodule adjacent to capsule; otherwise size, echogenicity, echotexture, capsular margins, and vasculature are unremarkable.
Left Kidney: Subjectively normal in size, normal in shape, and has adequate corticomedullary definition. No pyelectasia noted.
Right Kidney: Subjectively normal in size, normal in shape, and has adequate corticomedullary definition. No pyelectasia noted.
Left Adrenal: Normal size (0.68 cm at caudal pole); no nodules or masses noted.
Right Adrenal: Normal size (0.76 cm at caudal pole); no nodules or masses noted.
Urinary Bladder: Mildly distended with anechoic fluid. Normal wall thickness. Small amount sediment.
Stomach: Contains gas with shadowing and heterogeneously hyperechoic fluid with normal wall thickness and layering. The pylorus is not visualized.
Pancreas: Normal.
Small Intestine: Normal wall thickness and layering.
Large Intestine: Normal wall thickness and layering. The ICJ is not visualized.
Mesentery: Normal echogenicity; no free peritoneal fluid noted.
Lymph Nodes: The mesenteric, ileocolic, and medial iliac lymph nodes are normal.

Impressions

Liver - suspect possible vacuolar hepatopathy (endocrine vs. idiopathic vs. other) vs. nodular hyperplasia vs. combination vs. other.
Gallbladder - rule out forming mucocele vs. cholecystitis/cholangitis vs. incidental vs. other.
Spleen - consistent with benign myelolipoma.
Urinary bladder - sediment - rule out active vs. incidental.

Recommendations

1. If not already performed, recommend the following:

- current CBC/Chem/T4 to reassess liver values in particular as well as all other values.
- current/recheck UA with UCS given previous UA showed results supportive of UTI; if UCS negative and no other active sediment with proteinuria present, then recommend UPC.
- systolic BP
- adrenal function testing - despite normal adrenal glands on AUS, would recommend assessing adrenal function - typically ACTH stim or LDDST, although full Adrenal Panel can be considered which would also assess for possible atypical Cushing's disease.

2. Pending the above, consider FNA/cytology of liver +/- referral for cholecystocentesis. Cytology could at least help to rule out round cell disease (which is not highly suspected) and/or may help confirm VH and/or NH; however, ultimately surgical biopsies of liver and assessment of GB may be warranted to confirm and rule out other disease. Alternatively, at least at this time could consider VH +/- NH as most likely diagnosis pending serial bloodwork and US monitoring – see below.
3. Vacuolar hepatopathy is a significant possibility in this case (pending recheck bloodwork results) and may be present by itself or in combination with nodular hyperplasia.

- VH may be due to steroid hepatopathy, which could be due to Cushing's disease, although endogenous steroid treatment or chronic stress from illness (often unrelated to liver itself) can potentially cause this condition without true Cushing's disease being present. Dogs should have testing for Cushing's disease to rule it out as a cause for VH, even if as with this case the adrenal glands are normal or even decreased in size (or not clearly visualized). Since other concurrent diseases in other organ systems, as well as endogenous toxins, drug or supplement reactions, and/or nutritional issues could potentially cause VH, the patient may need to be assessed for cardiac disease, renal disease, neoplasia, dermatologic

disease, dental disease, immune and inflammatory diseases, and nutritional deficiencies and/or high-fat diet which could lead to obesity and to VH. Full assessment of history and current medications and supplementations should be performed as well.

- Many dogs with VH have an idiopathic cause. These would include patients in which the above causes have been ruled out, although there may be some overlap with hormonal disturbances other than typical Cushing's disease. Thankfully most dogs with VH retain normal liver function and ultimately do not experience development of significant liver disease or dysfunction from their VH. In some cases it may be elected to suspect VH without at least initially pursuing biopsies (if other causes/diseases are ruled out as discussed above); in this case, empirical treatment with Denamarin or similar liver protectants can be undertaken. However, even with likelihood of VH that may not cause overt liver dysfunction, if no biopsies are done initially, serial monitoring of bloodwork and (if warranted) recheck of liver appearance via US should be performed, and if significant further liver enzyme changes occur then biopsies should definitely be pursued at that point.
- There is an exception to the above in that Scottish Terriers are prone to a progressive form of vacuolar hepatopathy which can ultimately lead to liver dysfunction and disease. Other breeds may be susceptible so this should be kept in mind with monitoring and further diagnostics and treatment.
- In general the following should be recommended in cases of suspected or confirmed VH:
    - Testing to rule out Cushing's disease – ACTH stimulation test or LDDS testing.
    - CBC/chemistry; UA +/- UCS +/- UPC; T4
    - Full assessment via PE, history, and clinical signs to assess if there may be underlying disease in another organ system – including cardiac, GI, renal, dermatologic, and dental disease. In addition, if the patient is overweight/obese and possibly on a high-fat diet, this could contribute to VH and may need to be addressed with lower fat diet, weight loss plan, and if warranted treatment for hyperlipidemia if present.
    - If other underlying diseases/conditions are ruled out, treatment with Denamarin or similar sam-E (or n-acetyl cysteine based) supplement can be considered for liver health support. However, since most dogs do not develop significant liver disease or dysfunction from VH, treatment for the condition itself may not be warranted. It is important to remember that many dogs with VH never have resolution of elevated liver enzymes especially ALP.

4. If recheck bloodwork results are normal, consider serial monitoring of bloodwork +/- AUS findings.

At any time consider referral to a specialty facility for further diagnostics and treatment.

The information contained in this consultation is based on available imaging and data provided by the treating clinician. Assessments made and clinical thoughts are based on and may be limited by the quality of the data provided. The treating clinician remains responsible for determining the final treatments and applicability of the information herein. As clinical changes occur in patient status or clinical/laboratory values, the recommendations may need to be altered or removed.

Report on 2024-06-05 16:39:31 UTC signed by:

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