Signalment: Gidget, 12 year old, female spayed, Scottish Terrier, 10.7 kg

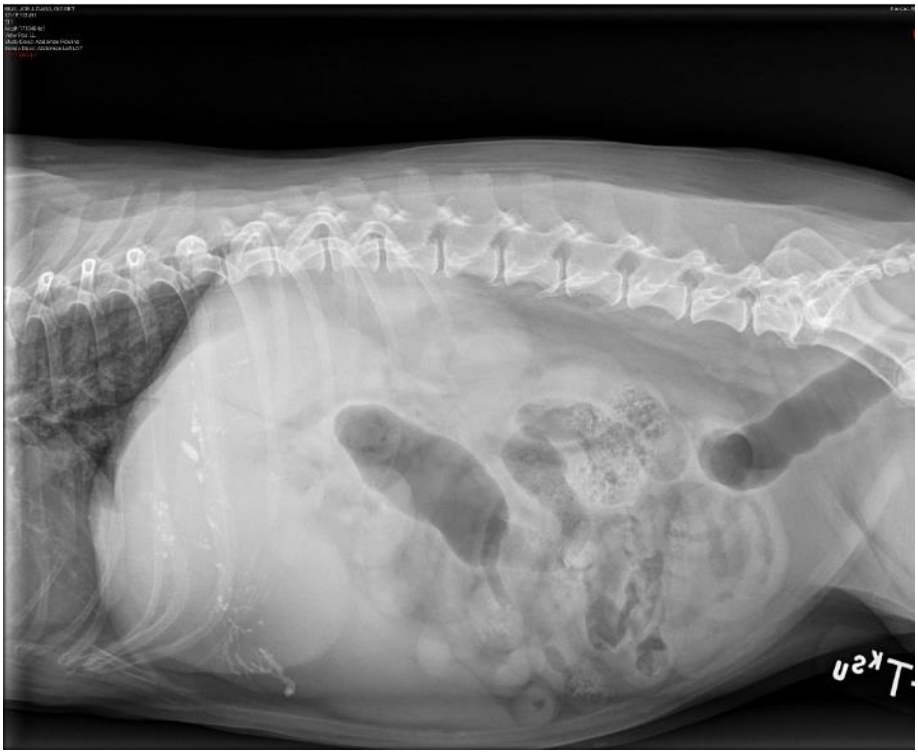
Presenting Complaint: Gidget presented after having elevated liver enzymes, patchy alopecia and PU/PD.

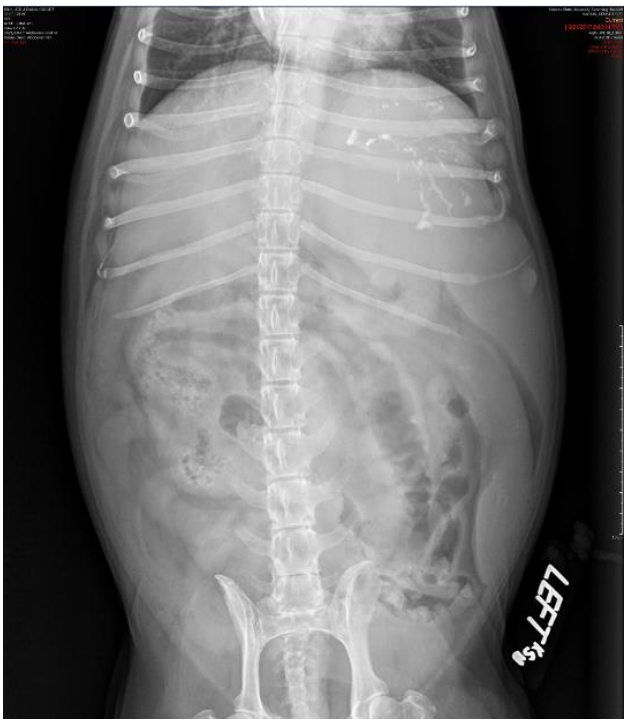
History: Gidget had been developing a patchy alopecia and pendulous abdomen as well as being PU/PD for a few months. At her regular veterinarian she had elevated liver enzymes, unremarkable abdominal radiographs and an unremarkable urinalysis. She was started on prednisone, Denamarin, and ciprofloxacin. After 3 weeks, her bloodwork was repeated and no changes in liver enzymes were noted which lead to her referral.

Physical Exam: Gidgit was bright, alert, responsive and had a normal TPR. She had alopecia along both flanks and a pendulous abdomen. On abdominal palpation, she had suspected cranial abdominal organomegaly. She had two small masses, one of her right hind foot on the 4th digit and another in her left ear.

Diagnostics performed:

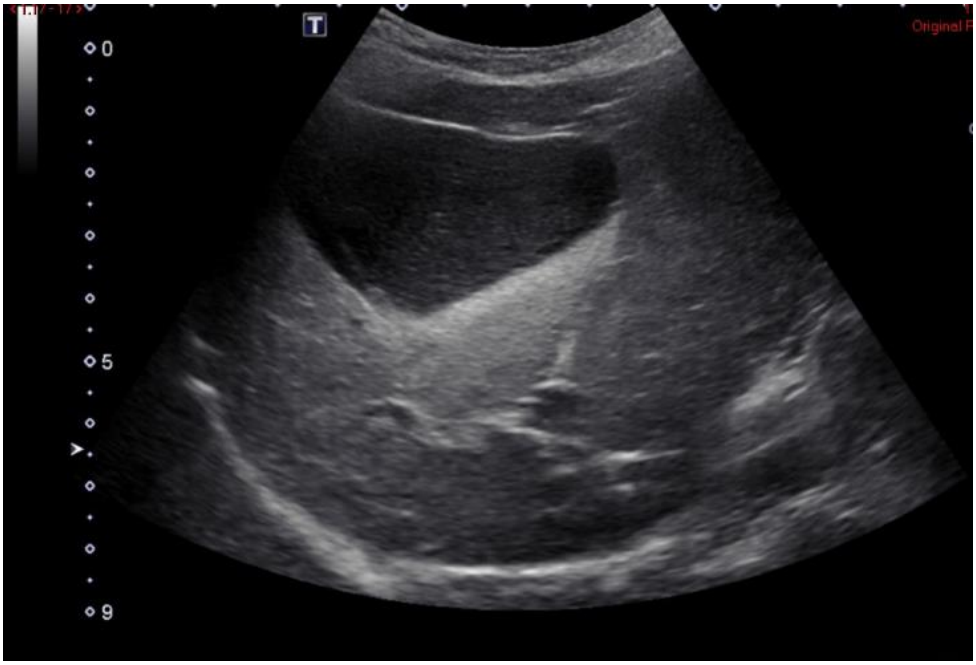
- **CBC**: unremarkable
- **Chemistry**: increased BUN (46), increased ALT (560), increased ALP (3625), increase cholesterol (544)
- **Urinalysis**: USG- 1.014, 3+ protein, urine culture showed no growth
- **Abdominal Radiographs**- **2 view abdominal radiographs**

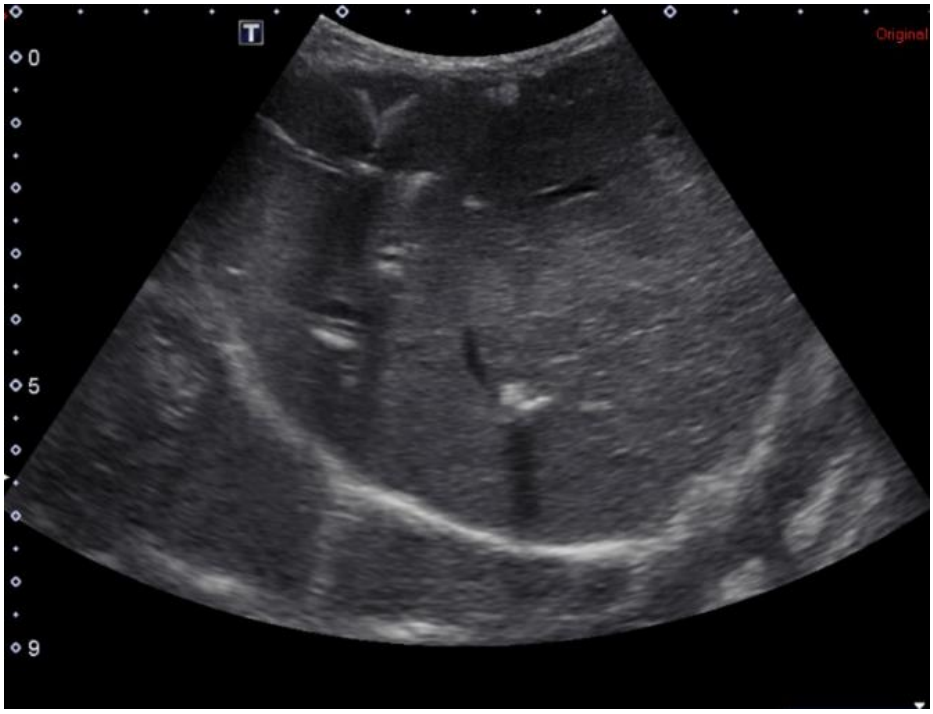




- **Findings**
  - Abdominal contour is pendulous
  - Branching mineral opacity superimposed on the left hepatic silhouette
  - Caudodorsal displacement of the gastric axis
  - Spleen displaced axially by liver lobes
  - Gas and soft tissues within the small intestines, gas within the colon
- **Interpretation**
  - The caudodorsal displacement of the gastric axis indicated hepatomegaly. Differentials for hepatomegaly include endocrine/metabolic changes (hyperadrenocorticism, vacuolar hepatopathy, etc.), benign hyperplasia, with lesser consideration given to neoplasia
  - The mineral opacity superimposed on the hepatic silhouette are most likely choledocholithiasis which is mineralization within the biliary tree. This is often an incidental fining but can be associated with neoplastic changes.

Abdominal Ultrasound:





- **Findings**
  - Moderately distended gallbladder with echogenic bile that is gravity dependent
  - Liver subjectively enlarged with rounding of the caudal ventral margin

        - Hypo to isoechoic nodules within the liver
        - Liner branching hyperechogenicities within the liver
        - Small cysts in the left kidney (largest measuring 5 mm)
        - Linear mineralization in the cortex of both kidneys
        - Left and right adrenals are normal in echogenicity, shape, and size
    - **Interpretation**
        - Hepatomegaly with isoechoic to hyperechoic nodules. Differentials for the enlarged liver include metabolic/endocrine changes (hyperadrenocortism, vacuolar hepatopathy, etc.), benign hyperplasia, with lesser consideration to neoplasia.
        - Choledocholithiasis and cholethiasis. These mineralization changes to the biliary tree and gallbladder respectively are most often incidental findings, but have the potential to lead to cholestasis.
        - Unremarkable adrenals
        - The cysts in the kidneys are indicative of degenerative changes most likely an incidental finding related to the patient's age.
- **Low Dose Dexamethasone Suppression Test**
    - Elevated cortisol at 4 and 8 hours post-dex administration consisted with hyperadrenocorticism

Discussion:

The two top differentials for Gidgit's hepatomegaly were hyperadrenocorticism and vacuolar hepatopathy ("Scottish Terrier Hepatopahty"). Scottish Terriers are predisposed to a breed related accumulation of glycogen resulting in a vacuolar hepatopathy. Dogs with this condition will have an elevated ALP and may show clinical signs similar to hyperadrenocorticm. However, they do not respond to treatment for hyperadrenocortism. Definitive diagnosis of this condition is made with a liver biopsy. It is important to monitor Scottish Terriers that have suspected vacuolar hepatopathy as they have been shown to have an increased risk for developing hepatocellular carcinoma. It is recommended that these dogs have regular blood chemistries and abdominal ultrasounds performed.

To rule in or out hyperadrenocorticism, a low dose dexamethasone suppression test was performed and Gidget had elevated cortisol at 4 and 8 hours post-dexamethasone administration, confirming a diagnosis of hyperadrenocortiscm. Gidget displayed many clinical signs often associated with Cushings including polydipsia/polyuria, having a pendulous abdomen and alopecia. Hyperadrenocorticm has two etiologies, a functional adrenal tumor secreting cortisol (ADH) or a pituitary tumor secreting ACTH (PDH) that stimulates the adrenal to secrete excess cortisol. In dogs with this disease, 85% have PDH and 15% have ADH. A LDDS test can be helpful to differentiate the two as 70% of PDH will suppress at 4 hours post-dex administration and escape that suppression at 8 hours. Abdominal imaging via ultrasound is another way to differentiate the two. A dog with PDH may have bilateral adrenal hypertrophy or bilaterally adrenals of normal size, while dogs with ADH would have unilateral enlargement and contralateral atrophy. However, not all adrenal masses are functional, so this has the potential to be misleading. Gidget's adrenals were symmetrical and normal in shape and size, suggesting a PDH. Gidget's ultrasound and blood chemistry also showed evidence of cholestasis. Cholestasis, including gallbladder mucoceles have been associated with PDH.

Gidget's ultrasound and radiographs also showed choledocholithiasis or mineralization of the biliary tree as well as choleliths within the gallbladder. Often these are incidental findings but they have the potential to cause biliary duct obstruction which would lead to clinical problems. Mineralization of the biliary tree has occasionally been reported in dogs with bile duct carcinoma. It is important to monitor these patients for clinical signs related to bile duct obstruction such as abdominal pain, lethargy, fever, jaundice, ascites, shock and systemic inflammation.

References:

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