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Patient Data			
Owner name	???????	Animal name	???????
Identification Exam Description	???????? ????????	Exam Date	??/???/2023
Performing Physician	S KFIR	Report Date	??/???/2023

Observations

Abdominal remarks

Anton was presented for abdominal ultrasound examination due to a long history of vomiting, poor appetite and colitis. A recent (month previously) episode of pancreatitis (confirmed with high specPL) was continuing with occassional vomiting, PUPD and poor appetite. No weight loss was noted.

Anton required sedation for the scan. Prior to the sedation, I palpated his abdomen; this was unremarkable and not painful. Anton was in slightly poor body condition (3/9 BCS). Anton had not been starved for the requested 12 hour period prior to the scan and there was significant gas and ingesta in his intestines due to this - it did not overall affect the scan but caused difficulties viewing some organs especially the right adrenal. No free fluid was present and the abdomen and contents were generally of normal echogenicity.

Liver was of normal size, well under the costal arch and with a sharp caudal border. It was of normal echoqunicity and echotexture.

The gall bladder was of expected size and position. The contents were mainly anechoic but a small amount of hyperehoic mobile sludge was seen taking up about one third of he gall bladder volume, non-shadowing and mobile when Anton was turned over. The lumenal side of the wall had multiple small hyperechoic projections into the lumen - these represent polyps. One luminal wall mass was much larger than the others, more hyperechoic (but non-shadowing) and situated at the neck of the gall bladder. The gall bladder was within expected size so obstruction is unlikely at this time. This mass could represent a polyp or neoplasia.

The spleen was generally normal in size and echogenicity and echotexture, though there was some hyperechoic patches around the mesenteric vein which likely represent myelolipoma, a common benign change of unknown significance at this time. The tail of the spleen also contained a solitary hypoechoic focal lesion of 4mm diameter - differentials include neoplasia (primary, metastases, lymphoma), nodular hyperplasia, and extramedullary haematopoesis. Biopsy would be required to differentiate these causes - an ultrasound guided FNA could be attempted and may give a diagnosis.

Most of the pancreas was visible to a normal degree, some hidden in the GIT gas shadowing, with normal echogenicity and normal echogencity of the surrounding fat.

The stomach was empty apart from gas so good images were obtained. The wall was generally normal apart from around the body of the stomach into the pylorus where the wall was mildly thickened but the muscularis layer was notably thicker than a normal stomach with preservation of the layers - this could represent chronic hypertrophic pyloric gastropathy or inflammatory condition/chronic gastritis. Pancreatitis can cause thickening of the stomach wall due to oedema - this is

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usually submucosal layer but can be muscularis (Murakami et al 2019). Full thickeness biopsies would be needed to differentiate the cause.

Several sections of small intestine (jejunum) were moderately thickened up to

6mm, most 4-5mm (normal for this BW 3.0 +/-0.5mm) - the thickedned sections varied in thickening and roughly accounted for about half of the jejunal loops visible. The layering was normal and the mesenteric lymph nodes were of normal size and echogenicity. Some of the sections of thickened jejunum the mucosal layer contained multiple small hyperechoic speckles - these are commonly seen with inflammatory bowel conditions. The colonic wall was of normal layering and thickness. The ileum was not visible due to GIT gas.

Both adrenals were visualised (the right with some difficulty and not completely clear image) and were bilaterally mildly enlarged - left adrenal 8.5mm diameter (normal for BW 6.4), right adrenal 7.8mm (7.5mm). They were uniformly hypoechoic. This may be due to hyperadrenocorticism (especially pituitary dependant), bilateral primary neoplasia or metastatic neoplasia. In my opinion the later two possibilities are less likely due to the uniform appearance of the glands and the relatively mild enlargement (some papers suggest neoplasia is more likely when the gland is >20mm diameter). No mineralisation was seen or invasion of the adjacent blood vessels. FNA of the adrenal glands an be undertaken and a minimally risky procedure. If HAC is suspected then further testing should be carried out.

Both kidneys were normal apart from a small (3mm diameter) anechoic cystic lesion in the cortex of the right kidney. This is likely an incidental finding and does not need further investigations with the lack of other ultrasound findings of the kidneys and normal renal blood tests.

Multiple abnormalities were found on Anton's scan and though ultrasound does not provide a cellular diagnosis of the causes, it can suggest where to target further investigations.

Ultrasound of the pancreas did not yield any positive signs of acute pancreatitis and Anton was comfortable on abdominal palpation prior to sedation. Ultrasound is insensitive for pancreatitis and pancreatitis, especially chronic, can be present with an entirely normal looking pancreas though an abnormal pancreas is highly specific for pancreatitis. Abnormal specPL levels can also be found with conditions other than pancreatitis such as IBD and GIT foreign bodies, and in hyperadrenocorticism (Mawby et al 2014).

The prescence of biliary sludge may indicate delayed gall bladder emptying and polyps are commonly found in inflammatory gall bladder conditions (cholangitis). The larger and more hyperechoic gall bladder wall mass could represent a polyp or neoplasia, though not currently causing obstruction - biopsy by FNA could be attempted along with cylecystocentesis (for cytology and culture) to investigate possible underlying chloangitis.

With the thickened stomach wall (in particular muscularis layer) and sections of the jejunum thickened and with mucosal speckling, a degree of inflammatory bowel disease is likely. This brings to mind the 'triaditis' syndrome in cats where biliary inflammation, gut inflammation and pancreatitis co-exist concurrently and I have come across dogs where I suspect this triad of diseases occurs too.

Anton has bilaterally enlarged adrenal glands. This could represent hyperadrenocorticism so this should be tested. A useful initial test is a urine creatinine:cortisol assay as if negative then he really can't have HAC (I recommend doing 3 samples over 3 days and waiting a good few days after any veterinary visit to reduce the chance of false positives). This could be followed up with an ACTH stim or low dose dex if positive.

Anton's spleen contained a single nodule which could be neoplasic or benigin (nodular hyperplasia or extramedullary ahematopoesis. An ultrasound-guided FNA

Conclusions

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could get a diagnosis for this or alternaltively, monitor by way of monthly ultrasounds.

Suggestions

- investigate if HAC is a possibility, especially as PUPD was reported
- if painful abdomen and pancreatitis is suspected then analgesia is a cornerstone of treatment oral paracetamol or injectable opiods
- Blood sample for cobalamin and folate as IBD seems likely, this will help with diagnosis and the guts will not heal without these being supplemented if deficient. The new range for cobalamin is >400ng/L.
- Hypoallergenic food trial is a crucial step and needs to be strict.
- +/- pancreatite enzymes to 'rest' pancreas during acute pancreatitis, though this has fallen out of favour.

SIGNATURE

Dr Sarah Keir BVMS PGCertSAM MRCVS RCVS Advanced Practitioner in Small Animal Medicine , ID:??????? ??/??/2023







6.9 mm

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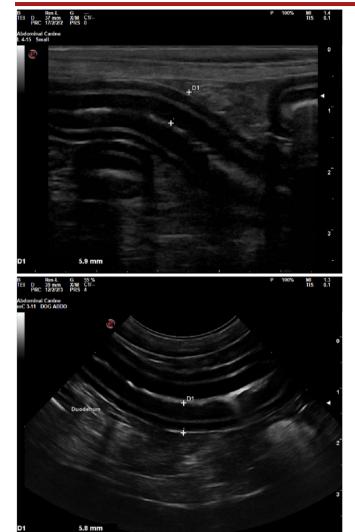








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