



PATIENT

Wagner Peppe

SPECIES

Canine

BREED

Poodle

SEX

Neutered male

AGE

6 years

WEIGHT

10 lbs

INTERPRETED BY

Eric Lindquist, DMV
DABVP, Cert. IVUSS

IMAGING PERFORMED BY

Dr. Wasserman

HOSPITAL NAME

Highlands AH

REFERRING VET

Dr. Frankenberger

INVOICE

75108

DATE

5/1/26

PRESENTING CLINICAL SIGNS

History: Prev hx of Brushite urinary stones (Jan 2024). Mineralized and enlarged prostate on x-rays taken 5/1/26. CBC and panel stable.

Abnormal PE/Chem/CBC/UA Results: See attached rads for supplemental review. Urinalysis via cysto pending today. Prostate FNA'd today with US guidance for culture and cytology by pathologist.

ULTRASONOGRAPHIC EXAMINATION OF THE ABDOMEN

Urinary System

The **urinary bladder**, trigone, and pelvic urethra presented normal thicknesses and normal tone. 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 and no evidence of pelvic dilation was present. The capsules were acceptably uniform without significant irregularities. The right kidney measured 3.96 cm.

The **prostate** was enlarged, irregular and mineralized measuring up to 1.7 cm. There was strong concern for prostatic carcinoma. Moderate blood flow to the prostate was noted.

Adrenal Glands

Both **adrenal glands** were 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 0.46 cm at the caudal pole and 0.36 cm at the cranial pole. The right adrenal gland measured 0.29 cm at the cranial pole and 0.35 cm at the caudal pole.

Spleen

The **spleen** presented a smooth homogeneous parenchyma hyperechoic to liver and renal cortical parenchyma. The capsule was smooth without noticeable expansion or deviation from within the spleen or adjacent pathology. The splenic vasculature demonstrated normal volume without signs of congestion or thrombosis. No sonographic evidence of acute or chronic inflammatory, neoplastic, or infarctual changes was noted.

Liver

The **liver** images submitted revealed subjectively normal liver size, contour, and structure. Parenchymal echogenicity was naturally coarse and hypoechoic to the spleen. Vascular and biliary tracts were of



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normal volume with no evidence of congestion. The gallbladder presented acceptably thin walls with primarily anechoic content. The cystic and common bile ducts were normal. No pathological hepatic lymphadenopathy was evident. No overt structural evidence of inflammatory, infiltrative or regenerative pathology was evident.

Gastrointestinal

Examination of the **gastrointestinal tract** revealed a stomach and intestine free of stasis, of normal wall thickness, acceptable curvilinear mural detail, and peristaltic activity. Small and large intestine demonstrated normal luminal chyme and stool consistency respectively. No obstructive or overt infiltrative disease was noted. No associated abnormal lymphatic activity was noted.

Pancreas

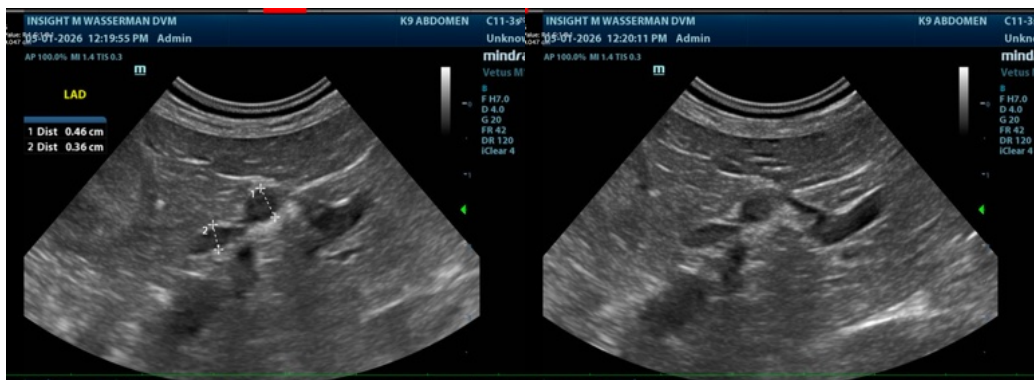
The base and limbs of the **pancreas** were observed to be largely isoechoic to surrounding omental fat. Pancreatic duct and capsular contour were acceptably normal and parenchyma respected normal curvilinear patterns. No overt evidence of active inflammatory or neoplastic disease was noted.

ULTRASONOGRAPHIC FINDINGS

Mineralized, enlarged prostate with positive vascularity. Strong concern for prostatic carcinoma.

INTERPRETATION OF THE FINDINGS & FURTHER RECOMMENDATIONS

Management is recommended based on cytology results. Eventual stent placement and chemotherapeutic intervention is recommended.





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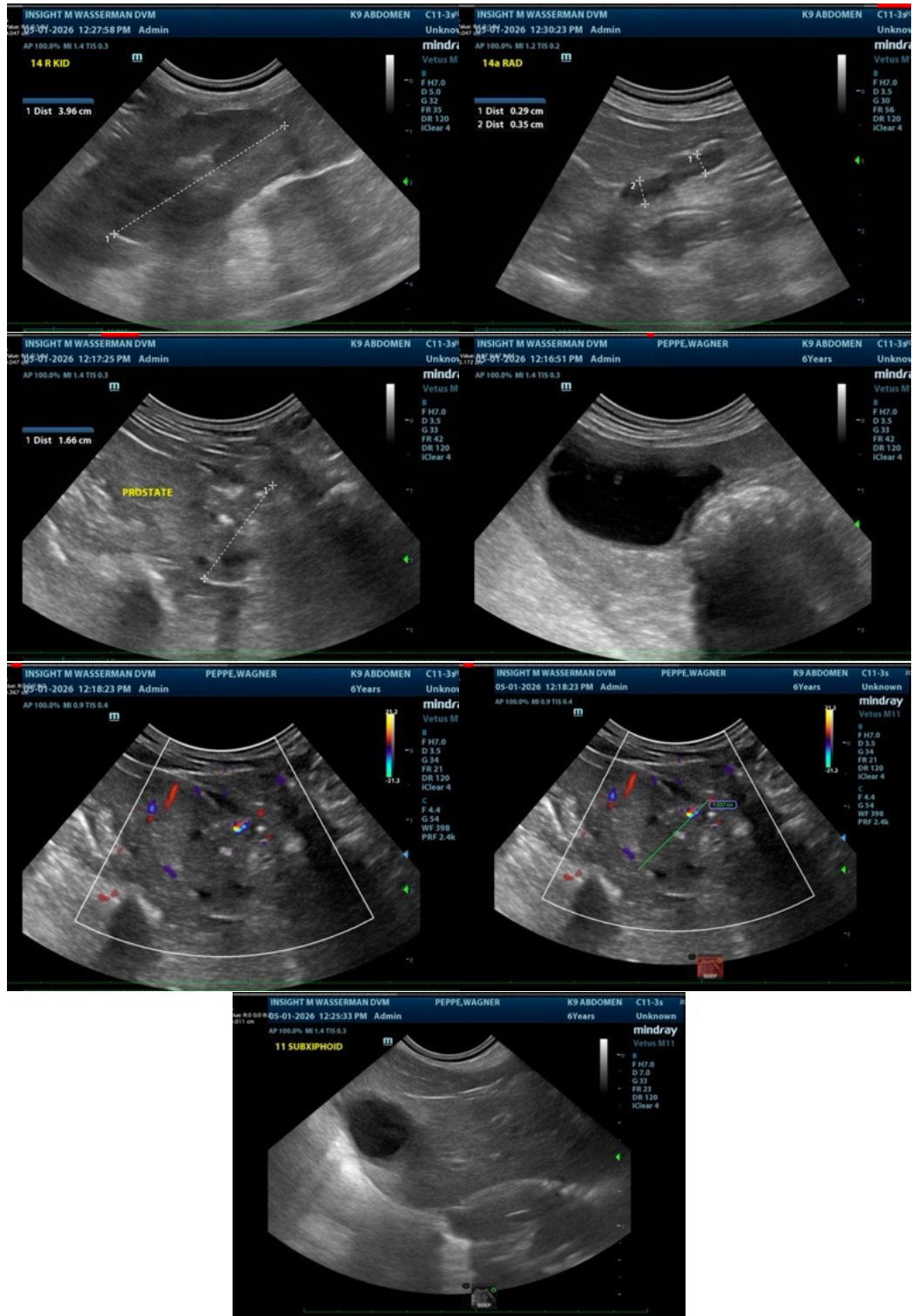
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The information and recommendations provided are based on the images presented by the

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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

Canine Prostatic Neoplasia

<http://www.sonopath.com/ProstaticCarcinoma>

Description: Prostatic neoplasia is frequently seen in dogs and can be diagnosed via ultrasonographic examination. The most commonly diagnosed prostatic neoplasms are adenocarcinoma and undifferentiated carcinoma. Transitional cell carcinoma (TCC) frequently spreads from the urinary bladder and urethra to the prostatic tissue (see the “Transitional Cell Carcinoma” chapter for more details). Metastatic squamous cell carcinoma, lymphoma, hemangiosarcoma, and leiomyosarcoma have been reported, but are less prevalent. Prostatic neoplasia has been documented in cats, but is quite rare.

Clinical Signs: Prostatic neoplasia presents in both neutered and intact males; however, a 2002 study suggested that neutered males were at greater risk for developing prostatic neoplasia than intact males. Typically, prostatic neoplasia is seen in older dogs (mean age of 10 years). Breed predilection includes mixed breed dogs, Shetland Sheepdogs, Dobermans, Scottish Terriers, and Airedale Terriers. Clinical signs and commonly reported signs from owners typically include: stranguria, frequent urinations, hematuria, dyschezia, weight loss, and decreased appetite. Other findings upon physical examination include fever, ataxia, pain upon rectal examination, and pain upon spinal palpation.

Diagnostics: Ultrasonographic examination should be performed if prostatic neoplasia is suspected. Common ultrasonographic findings include an enlarged, irregular prostate that typically has a hypoechoic appearance. Multifocal, poorly coalescing hyperechoic foci are also seen in prostatic malignancies. Hyperechoic foci are due to mineralization of the prostate; they cause far field shadowing. Cystic components can also be observed and are thought to indicate abscessation and/or necrosis. It can be difficult to differentiate chronic bacterial prostatitis from a prostatic neoplasia; however, regional lymphadenopathy is much more common with prostatic neoplasia than it is with chronic bacterial prostatitis. Malignancies of the prostate have often metastasized by the time of diagnosis. Frequent sites of metastases include the sublumbar lymph nodes, the pelvis, lumbar vertebrae, and the lungs. If metastases to the pelvis or lumbar vertebrae have occurred, bony lysis will often be noted radiographically. Metastasis to the liver, brain, kidney and spleen may



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occur. A definitive diagnosis of a prostatic neoplasm can be achieved through biopsy as well as fine needle aspiration (FNA) or through ultrasound-guided traumatic catheterization.

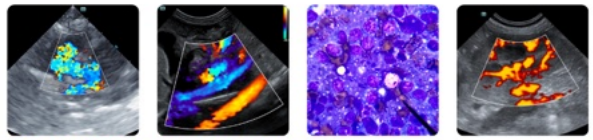
A complete and thorough workup includes a CBC, biochemical profile, urinalysis, as well as three radiographic views of the thorax, an abdominal ultrasound, and an ultrasound-guided prostatic biopsy or FNA, if indicated. Urinalysis may reveal hematuria and pyuria. Prostatic fluid analysis can also be helpful in identifying neoplastic cells.

Treatment: Unfortunately, once diagnosed, prostatic carcinoma offers a poor prognosis; prostatectomy, chemotherapy, and radiation therapy have proven unsuccessful in improving quality or length of life. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as deracoxib, meloxicam, and piroxicam, have been used for their palliative, anti-neoplastic properties with prostatic carcinomas. Certain tumors, including various carcinomas (e.g. TCC, prostatic carcinoma, mammary carcinoma, squamous cell carcinoma) overexpress COX-2, which converts arachidonic acid to prostaglandin G2 (PGG2)/prostaglandin H2 (PGH2), and ultimately to prostaglandin E2 (PGE2). The metabolite, PGE2, is associated with increased inflammation, tumor invasiveness, angiogenesis, and reduced apoptosis. In vivo and in vitro, NSAIDs inhibit COX-2, resulting in the suppression of PGE2, and thereby inhibiting tumor growth and metastasis. This effect has been achieved with both non-selective COX inhibitors as well as COX-2 inhibitors (the latter will suppress COX-1 at increased doses).

Some cases of prostatic carcinoma are managed palliatively with cyst/abscess ultrasound-guided drainage, antibiotic infusion, systemic antibiotics, and NSAID treatment and/or chemotherapy. Anecdotally, it has been observed that patients that often present clinical signs of hematuria or dysuria owing to cyst or abscess formation may be treated with repeat ultrasound-guided drainage. This appears to work especially well if there is a considerable cystic component to the prostatic tumor. The key is to image the prostate adequately, drain any cysts that are present, sample the abnormal parenchyma (FNA or biopsy), and potentially infuse antibiotics directly into the cystic cavities if a suppurative fluid is retrieved. The patient should be monitored clinically over time and reevaluated to see if cysts recur. Every case responds differently to treatment, and the behavior of parenchymal and cystic growth will vary.

Currently, investigational studies involving fluoroscopic-guided direct chemotherapeutic embolization through the iliac arteries as well as urethral stent placement are offered by select tertiary veterinary facilities that have an interventional radiology department. Ultrasound-guided endoscopic diode laser ablation through a perineal urethrostomy is also being attempted as a salvage and palliative procedure.

Conclusion: Prostatic neoplasia is more commonly detected in neutered male dogs than intact males. Diagnosis is typically obtained using ultrasound, cytology, and histopathology. Unfortunately, traditional therapy typically yields a guarded to poor long-term prognosis, but palliation with NSAIDs



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and/or chemotherapy can temporarily improve clinical signs. Investigational techniques may provide additional therapeutic options but are currently experimental.

References:

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