Canine Imaging Self Assessment: Acute onset vomiting in a Tibetan Terrier

Signalment and History

A 3-month-old female entire Tibetan Terrier was presented for investigation of acute onset vomiting and lethargy. The patient had experienced vomiting for 48 hours, which was also associated with an episode of diarrhoea. The owner had noted poor body growth, poor weight-gain and suboptimal body condition despite demonstration of a good appetite prior to her presenting complaint. She had no associated clinical signs of polydipsia or polyuria at home. To the owner's knowledge there has been no access to any known toxin, or evidence of ingesting foreign material that could result in gastrointestinal obstruction, but halitosis had been noted for approximately 8 weeks. She was fully vaccinated and had been treated routinely for endo- and ectoparasites.

Clinical examination

On presentation, the dog was lethargic but was responsive with a normal body temperature. There was an increased respiratory rate (60 breaths per minute) without dyspnoea. The mucous membranes were pink, without evidence of congestion and with a capillary refill time of <2seconds. The heart rate was 180 beats per minute with normal pulse quality. On auscultation of the thoracic cavity, the lungs demonstrated increased respiratory noises with pulmonary crackles. Abdominal examination was unremarkable.

Survey radiographs of thorax and abdominal were obtained in right lateral recumbency to screen for causes of acute vomiting (Figure 1).

Describe the main findings on the radiograph. Based on the findings construct a differential diagnosis list.

Figure 1 is a right lateral projection of the thorax and abdomen. There are thin, linear, radio-opacities that appear to be contained within the gastric wall – these are suggestive of mineralisation of the rugae. There is a mild reduction of the peritoneal serosal detail in the patient - this can be commonly observed in a young patient due to the brown fat deposits within the abdomen. The peritoneal definition can be altered in patients with free abdominal fluid – there is no evidence of this in this case. The vertebrae and both femurs demonstrate decreased bone opacity radiographically, and there is thinning of the cortices of the distal portion of the femur giving a double cortical sign seen with osteopenia; this is due to intracortical bone resorption seen with cases of nutritional secondary hyperparathyroidism, suspected renal secondary hyperparathyroidism. There is evidence of loss of the fine trabeculation of the vertebral bodies, spinous processes and the distal portion of both femurs, leaving a coarse pattern and generalised loss of bone density. On assessment of the thoracic cavity, there is a diffuse opacity affecting the lung fields; this appears to be an interstitial lung pattern. The thoracic findings are likely to normal for age of the dog; this is thought to be of no clinical significance and was not further investigated.

Key radiographic findings:

- Mineralisation within the stomach, suspected gastric mucosal mineralisation.
- Generalised osteopenia affecting the long bones and vertebrae.

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Differential diagnosis:

- **Primary Hyperparathyroidism**
- **Secondary Renal hyperparathyroidis**m
- **Uraemia (**uraemic gastropathy) secondary to renal failure may be suspected due to the tissue mineralisation of the gastric wall.
- **Gastric foreign body (chronic)** – this was thought to be less likely but a radio-dense foreign body may be considered.

What other investigations should be considered?

In this case, with the clinical manifestations, signalment, and the radiographic findings, there was a suspicion of chronic renal disease (secondary dystrophic calcification of the gastric wall with radiographic evidence of osteopenia). Due to the systemic signs noted routine biochemistry and haematology were performed. The results of the bloods are shown in Table 1 (haematology) and Table 2 (serum biochemistry).

The haematology revealed a mild anaemia that appeared poorly regenerative in nature (normocytic (normal mean cell volume [MCV]), normochromic (normal mean cell haemoglobin concentration [MCHC]) and low to normal reticulocyte count). The biochemistry results showed a marked elevation in the serum urea (BUN) and creatinine levels. There was hyponatraemia and hypocholaemia noted. The electrolytes abnormalities could be associated with excessive gastrointestinal losses (including vomiting Cl- rich stomach contents) or through excessive urinary losses.

Potential causes of azotaemia:

1. **Pre-renal Azotemia:** In this circumstance there is reduced renal perfusion due to low blood volume. There may be accelerated production of nitrogenous waste products (e.g. BUN) because of enhanced catabolism of tissues in association with infection, fever, trauma, or the use of corticosteroids . BUN can become elevated with gastrointestinal digestion and absorption of protein sources- this can include a high-protein diet or gastrointestinal hemorrhage.
2. **Renal Azotemia:** Acute or chronic kidney conditions that impair at least seventy-five per cent of the renal function can result in an ensuing azotaemia.
3. **Post-renal Azotemia:** The azotaemia is usually the result of altered urine excretion such as urinary obstruction or rupture of the urinary tract. Urinary clearance and excretion of the nitrogenous products is altered.

Additional ancillary tests that would be indicated include urinalysis, blood gas analysis, serum phosphate, and serum calcium levels (total and ionised), to further identify the underlying pathology. Urinalysis acts as a basic, inexpensive and quick diagnostic tool, and can evaluate for renal and non-renal causes of azotaemia; urine specific gravity can facilitate the assessment of kidney function and concentrating ability of solutes.

Which imaging modality would you use next and what would you be looking for?

Abdominal ultrasonography was performed to assess the renal architecture and shape, and allow further assessment of the gastrointestinal tract. Still ultrasonographic images are shown of the stomach (Figure 2a), left kidney (Figure 2b) and right kidney (Figure 2c). Additional to the abdominal imaging, a dorsoventral radiograph was

taken of the patient's skull (Figure 3).

What are the major abnormalities on the sonographic images? What additional information is available from the radiograph of the skull?

The stomach wall (Figure 2a) appears to be thickened, with hyperechoic mucosa. The images are consistent with inflammation and mineralisation affecting the stomach wall - this is most likely due to uraemic gastritis. There was no suggestion of a gastrointestinal foreign body on assessment of the gastrointestinal tract; there was an empty stomach without shadowing from a foreign interface, while the small intestines were within normal limits without a gravel sign.

The ultrasound images of both kidneys (Figures 2b and 2c) show a generalised increase in echogenicity and a loss of the normal renal architecture; there is poor corticomedullary differentiation. The kidney parenchyma is difficult to identify when compared to the surrounding tissue, and these changes indicate chronic or dysplastic renal changes. The kidneys were not measured at the time of the abdominal examination – this may be useful as dysplastic kidneys are usually smaller on ultrasonography.

The skull radiograph (Figure 3) was taken to assess for loss of the lamina dura. There is loss of the lamina dura surrounding the alveolus. There is generalised reduction in the bone density, with loss of alveolar bone and ill defined lucency around the teeth of the maxilla and mandibular dental arcades (the "floating teeth" appearance) giving a generalised, coarse, lace like trabecular bone pattern. The cortices appear irregular – this is most marked at the zygomatic arch and the coronoid process. The radiographic changes are suggestive of secondary renal hyperparathyroidism; the loss of the lamina

dura is an early radiographic sign, followed by generalised loss of bone density of the skull and long bones.

What is the presumed diagnosis for this case?

The young age combined with the clinical findings on bloods and imaging is suggestive of a juvenile nephropathy; the most likely diagnosis is renal dysplasia resulting in progressive chronic renal failure, with secondary renal hyperparathyroidism and uraemic gastritis.

Discussion and outcome.

Renal dysplasia (RD) should be considered as a differential in juvenile patients with renal disease that exhibit an appropriate history, signalment and clinical findings, especially patients that have had no exposure to toxin ingestion. RD is thought to have a strong familial link with certain pure-breeds showing a predisposition, including but not limited to Boxers, Cocker Spaniels, Chow Chow, Golden Retriever, Lhasa Apso, Samoyed, Shih Tzu and Soft Coated Wheaten Terrier**.** It is a congenital and developmental malformation of the kidneys, which results from poor differentiation of the renal tissue, with the progressive manifestations associated with chronic kidney disease**.** The age of onset of clinical signs in congenital nephropathies ranges from a few weeks to several years of age, however many can remain quiescent until later in life**.** The clinical manifestations can vary in each patient, with the most common presenting signs being a reduced appetite, stunted growth or weight loss, polyuria and polydipsia, and varying gastroenteric signs.

Often affected dogs appear healthy and exhibit good urine concentrating ability initially. Proteinuria often develops followed by a reduced growth rate and reduced

urine concentrating ability. Subsequently serum concentrations of urea and creatinine progressively increase. Additionally, such patients will have other clinical findings associated with the chronic renal insufficiency: non-regenerative anaemia, azotaemia, hyperphosphataemia, hypokalaemia and a metabolic acidosis. Calcium levels are variable in these patients and may demonstrate low (may relate to low albumin levels), normal or high levels depending on the stage of the disease. The urine specific gravity is commonly isosthenuric (1.008 – 1.012) in such patients despite the animal showing azotaemia.

Ultrasound proves to be an effective tool to assess the kidney structure, giving supportive evidence for the clinical suspicions and facilitating the formulation of a concise differentials list based on the imaging findings. The ultrasound findings of renal dysplasia have been documented in dogs**.** The ultrasound appearance of affected kidneys may reveal thinning of the renal cortex with increased echogenicity or poor corticomedullary definition when compared to normal structure, with diffuse increased echogenicity of the parenchyma. There may be notable distortion affecting the renal pelves in affected patients and the kidneys are usually bilaterally reduced in size. Ultrasound alone cannot differentiate RD from other causes of fibrotic, end stage kidneys, although the age of the patient and other historical findings will usually indicate the likely cause.

When uraemia occurs, a degenerative, ulcerative gastritis can occur with consequential mineralization of the gastric mucosa and lamina propria (uraemic gastropathy) as seen in this case. Calcium homeostasis becomes altered in many patients suffering from renal failure due to acidosis, early hyperphosphatemia,

secondary parathyroid hyperplasia and poor synthesis of 1,25 dihydroxycholecalciferol by the diseased kidneys. Calcification of the tissues appears to be due to a combination of ischaemia, tissue degeneration and altered plasma concentrations of calcium and phosphorus.

Renal dysplasia cannot be definitively diagnosed from imaging or bloods alone, but requires histological examination of the renal tissues following biopsy or at necropsy (although the presentation and sonographic findings will often raise a very high index of suspicion).

There is no specific treatment for this condition and in clinically affected dogs showing with polydipsia, polyuria and uraemia, it continues to end-stage renal disease. The prognosis in this condition is very poor. These animals often decline rapidly despite supportive treatment; the poor quality of life often results in euthanasia. This dog was sadly euthanised due to advancement of the progressive clinical manifestations.

Further Reading

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222 **Figure Legends**

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224 Figure 1: Right Lateral Recumbency radiograph of the caudal thorax and abdomen.

Figure 2a: Long-axis ultrasonogram of the body of the stomach

Figure 2b: Long-axis ultrasonogram of the left kidney

Figure 2c: Long axis ultrasonogram of the right kidney

Figure 3: Dorsoventral radiograph of the skull.

Tables:

Table 1: Haematology

Parameter	Value	Reference Range
RBC (x1012/l)	**4.2**	5.65 - 8.87
HCT (%)	**25.8**	37.3 - 61.7
Hb (g/dl)	**9.7**	13.2-20.5
MCV (%)	**61.4**	61.6-73.5
MCHC (%)	37.6	32-37.9
Reticulocytes (K/uL)	38.6	10-110
Platelets (K/ul)	241	148-484
WBC (x109/l)	8.88	5.05 - 16.76
Neutrophils (x109/l)	5.46	2.95- 11.64
Lymphocytes (x109/l)	2.75	1.05 - 5.10
Monocytes (x109/l)	0.43	0.16-1.12
Eosinophils (x109/l)	0.24	0.06-1.23
Basophils (x109/l)	0	0-0.1

Abnormalities are highlighted in bold

Table 2: Serum biochemistry

Parameter	Value	Reference Range
Glu (mmol/l)	6.12	4.28 - 8.34
BUN/ Urea (mmol/l)	**46.4**	2.5 - 10.4
Creat (mmol/l)	**289**	27 - 106
TP (g/L)	54	48 - 72
Alb (g/L)	28	21 - 36
Glob (g/L)	26	23 - 38
Alb:Glob		
ALT (U/l)	**155**	8-75
ALKP (U/l)	121	46 - 337
Na(mmol/l)	**138**	145 - 157
K (mmol/l)	5.2	3 5 - 5.5
Cl (mmol/l)	**98**	105-119

Abnormalities are highlighted in bold