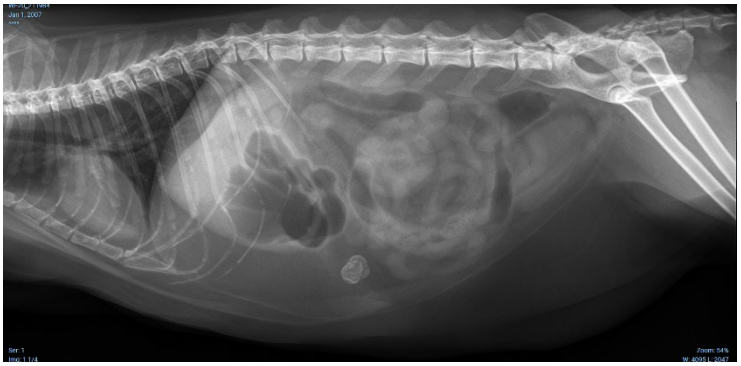
Hematology/biochemistry/coagulation profile

There was a moderate leukocytosis with moderate neutrophilia and with band neutrophils. Lymphopenia and eosinopenia were recognized. In the biochemistry profile, there was a marked hyponatremia, a mild hypoglycemia and hypochloridemia, a moderate hypercalcemia (ionized calcium) and a severe azotemia and hyperbilirubinemia present. Regarding the coagulation parameters, there was an elevated aPTT.

X-ray/sonography



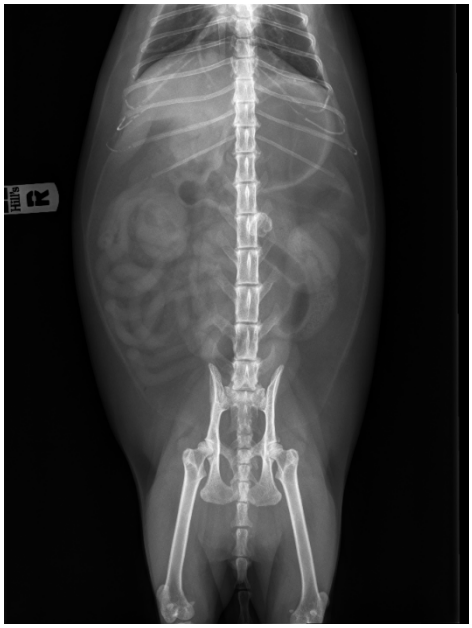
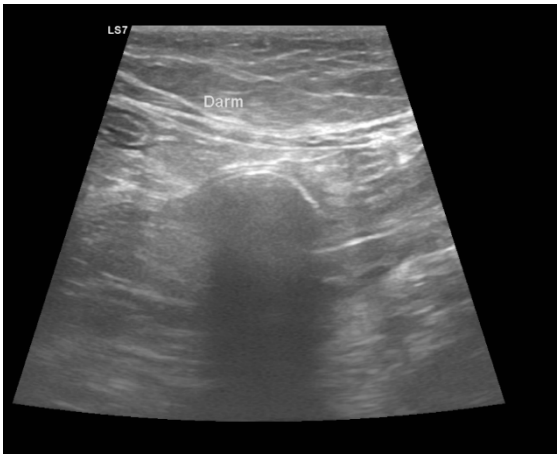


Figure 1: X-ray in laterolateral (left figure) and ventrodorsal projection (right figure) in a 13 years old European Shorthair cat presented due to vomiting and inappetence



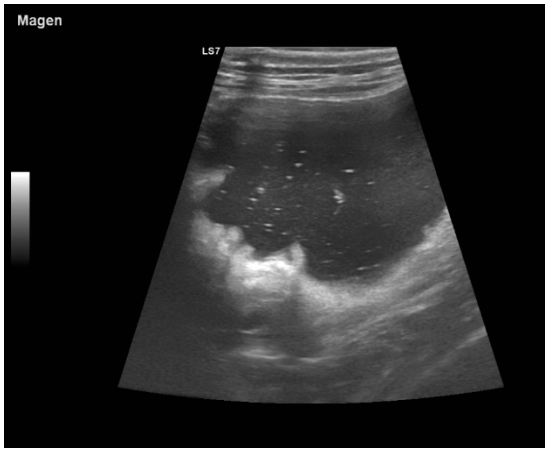


Figure 2: Ultasonography in a 13 years old cat presented due to vomiting and inappetence. The left figure shows a stomach filled with water, the right figure a foreign body in the small intestines with typical caudal disappearance of sound

The radiographic as well as ultrasonographical findings were highly suspicious for a foreign body in the small intestines. There was also evidence of an inflammatory condition with pancreatitis and small amount of free fluid in the abdominal cavity.

Diagnosis

- Foreign body (nut) in the small intestines
- Shock / inflammatory intestinal condition / pancreatitis
- Hypertrophic cardiomyopathy (HCM) diagnosed by sonography of the heart
- toxicity/ neurotoxicity probably from eating “trail mix”

Discussion

Shock is defined as a state, in which profound and widespread reduction of effective tissue perfusion leads first to reversible, and then if prolonged, to irreversible cellular injury.[1] There are six possible categories: obstructive disorders, metabolic disorders, Hypoxia, Hypovolemia, cardiogenic disorders and distributive/vasodilatatory disorders.[1] Cardiogenic shock was less likely as it is most often recognized in dogs with Dilatative Cardiomyopathy (DCM) as well as hypoxemic condition (i.e. due to anemia, pulmonary disease, methemoglobinemia), metabolic (i.e. toxic shock, severe hypoglycemia) and obstructive (i.e. due to cardiac tamponade, neoplasia) conditions. Hypovolemic shock due to profound dehydration was possible as well as distributive shock due to toxicity and/or sepsis/SIRS. There was evidence for inflammatory reaction in the pancreas region by ultrasonography and elevated DGGR-lipase, most likely secondary due to the foreign body in the small intestines and hypovolemia.

The moderate leukocytosis was most likely caused by inflammatory conditions due to the obstruction of the small intestines by the nut. This is supported by the borderline banded neutrophilic granulocytes. Eosinophilia and lymphopenia were suspicious for a stress response. Additionally, there was an elevated PTT in combination with a normal PT, making early DIC the most likely differential diagnosis.

Regarding the azotemia, there are three possible underlying conditions: prerenal, renal and postrenal.[3] Prerenal was the most likely differential diagnosis due to hypovolemia with lowered GFR, decreased cardiac output and shock. A renal involvement was also discussed, mainly because of the uptake od raisins containing toxins possibly leading to toxicity with kidney failure and probably also liver damage. Postrenal causes as i.e. urinary tract obstructions/ruptures were excluded due to the fact, that the cat was able to urinate.

The cat showed hyponatremia during first presentation. The cat showed clinical signs of dehydration, a condition normally seen with hypernatremia. However, possible differentials regarding the hyponatremia in this case include excess of water, endogenous shifts and/or sodium-deficits and reduced uptake from the GI channel.[4] In this case the obstruction of the small intestines probably contributes to the loss of electrolytes including sodium and chloride (from both NaCl and HCl) and furthermore the lowered uptake of hydrogen may also contribute to the development of the metabolic alkalosis in this case. Additionally, renal losses and losses due to vomiting had to be taken in consideration.

The differentials for the severe hyperbilirubinemia include prehepatic, hepatic and posthepatic disorders.[5] Prehepatic disorders seems less likely with hematology parameters in physiological range without anemia, making hemolysis unlikely. Hepatic disorders could be linked to the toxic and septic condition and additionally occur as a consequence of the obstruction of the small intestines. Furthermore, hepatocytic insufficiency could develop with oxygen deficiency (in this case because of shock and poor blood circulation), and the increase in S-Bilirubin could be secondary to reduced uptake and conjugation in dysfunctional liver cells. It should also be noted that among the enzymes the AST is higher than the ALT (with only minor increase) and normal ALP. The results could indicate a general toxicity to different organs including muscular and intestinal tissue (AST), rather than a specific liver cell damage (ALT show only a minor increase). The lack of response in ALP further support the hypothesis of other causes of liver insufficiency and elevation in bilirubin than a cholangio-hepatiitis with stasis/obstruction of the bile ducts. Posthepatic disorders can however not be excluded due to intestinal inflammatory conditions / pancreatitis.

Regarding the acid-base-disturbances, a metabolic alkalosis with partially compensatory respiratory acidosis was most likely due to the high ileus caused by the obstruction in the small intestines. There was also an increased breathing rate supporting this classification. Other explanations could be a lowered oxygen saturation of the blood due to the hypovolemic shock, reduced blood circulation resulting in a mixed condition and/or cardiomyopathy.

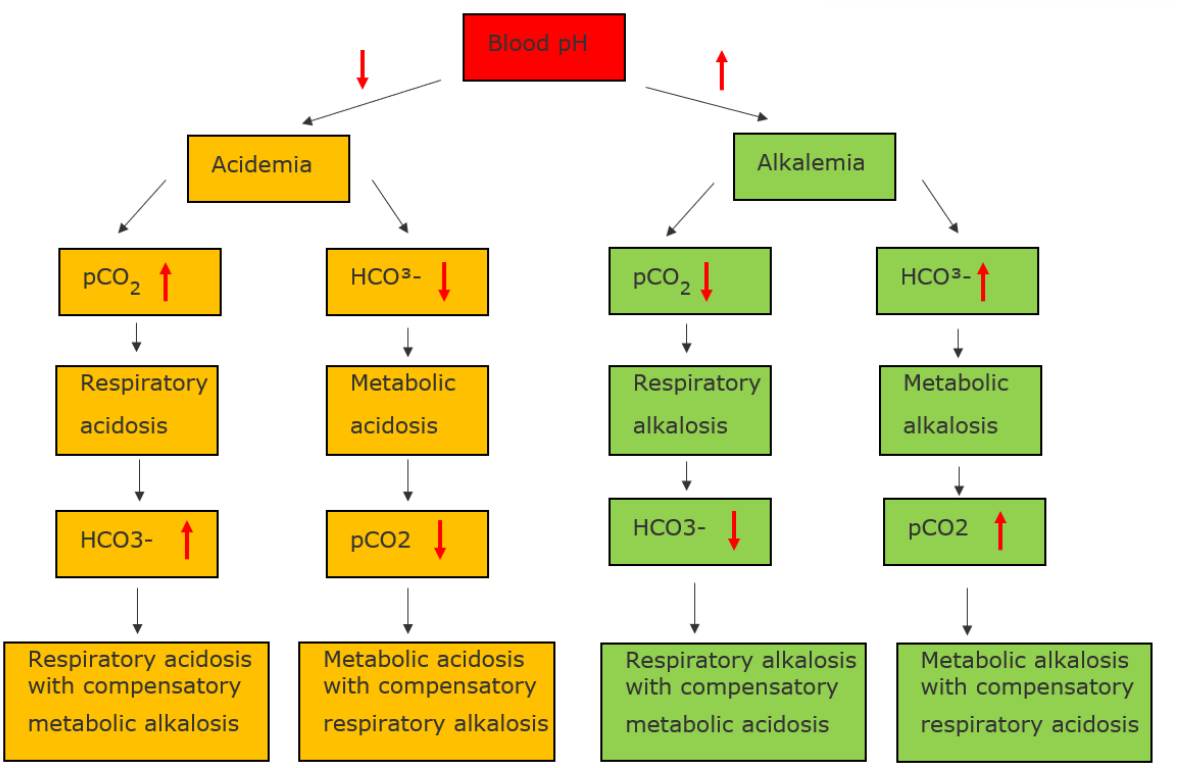


Figure 3: Algorithmic approach to classification of simple (not mixed) acid-base disorders (Stockham SL and Scott MA: Fundamentals of veterinary clinical pathology, chapter 10, page 579, Fig. 10.5)

Therapy/Outcome

The prognosis was guarded to poor due to suspicion of toxicity, risk for severe renal failure and liver insufficiency. The cat was stabilized over night and underwent surgery the next day, removing a nut out of the small intestines. Before surgery, sonography of the heart was done diagnosing a hypertrophic cardiomyopathy (HCM). During surgery, a feeding tube was placed into the oesophagus. After surgery, the cat was treated with i.v. infusion and heart medication, amoxicillin-clavulanic-acid (12.5 mg/kg bodyweight q12h i.v.), omeprazole (1 mg/kg bodyweight q12h i.v.) and maropitant (0.1 ml/kg bodyweight i.v.). After infusion therapy, the creatinine decreased from initially 530 µmol/L to 205 µmol/l on day 18. Unfortunately, after i.v. infusion was stopped, the creatinine raised again to 420

µmol/L on day 45. The cat recovered very slowly, there was no food intake and the owner applied food by the oesophageal tube. Because of the bad recovery and the severe azotemia, the owner decided to have the cat euthanized.

Conclusion

Intoxication from toxic food components, cardiomyopathy, intestinal obstruction and inflammatory condition of the pancreas. Metabolic alkalosis.

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