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Vet Record

ORIGINAL RESEARCH

Radiological and laboratory prognostic parameters for gastric dilation in rabbits (*Oryctolagus cuniculus*)

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Abstract

Background: The aim of this study was to evaluate the radiographical and laboratory findings in pet rabbits with gastric dilation (GD) and identify prognostic parameters.

Methods: One hundred and fifty-five rabbits with GD were included for the radiographical analyses and 75 for the laboratory analyses. A vertebral stomach score (VSS) was established to determine the extension of the stomach on lateral radiographs. In addition, the caudal limit of gastric distention and the extension and position of gas accumulation in the stomach were recorded.

Results: The VSS was significantly higher in rabbits with GD than in healthy rabbits ($p = 0.001$). Extensive amounts of gas accumulation in the stomach ($p = 0.001$) and a ventral or central location of gas accumulation ($p = 0.023$) were associated with significantly increased mortality. Azotemia (51%), hyperglycaemia (44%) and hyponatraemia (37%) were the most frequent biochemical alterations in rabbits with GD.

Limitations: Due to the retrospective nature of the data, the cause of GD could not be determined in all animals.

Conclusion: Rabbits with GD and severe alterations in glucose, creatinine and sodium concentrations, ventral or central gas accumulations and a large amount of gas in the stomach had a poor prognosis.

KEYWORDS
azotemia, gastric dilation, hyperglycaemia, hyponatraemia, rabbit, radiography

INTRODUCTION

Gastric dilation (GD) is an acute, often fatal, gastrointestinal disorder commonly affecting pet rabbits.[1,2] GD is frequently caused by intestinal obstruction, with pellets of compressed hair inducing 75-81% of these obstructions.[1–4] Other causes include beans and seeds (food components),[1,3] dried food material,[3] neoplasia,[1] synthetic material,[1,3] (postoperative) adhesions,[1,3] tapeworm cyst,[1] hernia,[1] omental entrapment[3] and diverticulosis.[1] GD is a radiographic finding characterised by an enlarged, fluid and gas-filled stomach ('fried egg stomach').[1,4] Dilated gas and/or fluid-filled small intestinal loops may also be present.[1–3] Only one study has described the radiographic features of GD in detail. This retrospective study by Debenham et al.[3] included rabbits that had radiographic evidence of intestinal obstruction and were subsequently treated surgically. Stomach size, as well as stomach and small and large intestine contents, were evaluated on radiographs and compared with those of healthy animals with no signs of gastrointestinal disease.

Two previous studies have investigated laboratory parameters in rabbits with GD. Harcourt-Brown and Harcourt-Brown[5] reported hyperglycaemia in rabbits with GD, and animals with glucose concentrations above 20 mmol/L often died. Meanwhile, Brezina et al.[4] found acidosis in 57% of rabbits with GD.

The aim of our study was to evaluate both the radiographic and laboratory findings for pet rabbits with GD and identify potential prognostic parameters.

MATERIAL AND METHODS

Population

The study included 155 pet rabbits presented to the authors' veterinary clinic over a period of 6 years.

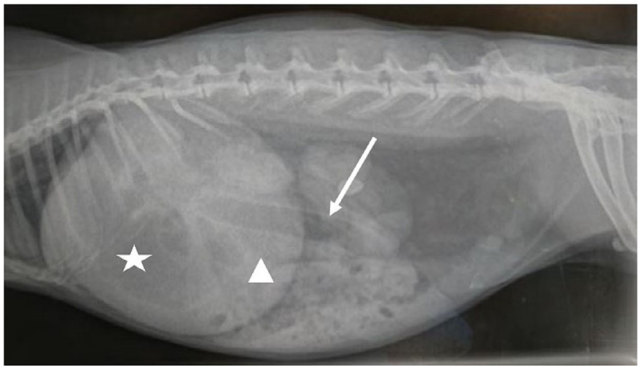

FIGURE 1 Right lateral radiograph of the abdomen of a rabbit with gastric dilation. The stomach is gas (star) and fluid (triangle) filled. A gas-filled dilated loop (arrow) of intestine runs across the stomach

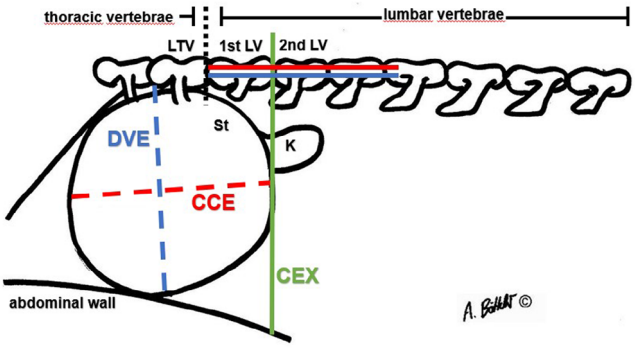

FIGURE 2 Schematic illustration of the measurements for the vertebral stomach score on lateral abdominal radiographs in rabbits. The craniocaudal extension of the stomach (CCE; red) and dorsoventral extension of the stomach (DVE; blue) are transposed onto the lumbar spine. CEX, caudal stomach extension; K, kidney; LTV, last thoracic vertebra; LV, lumbar vertebra (1st and 2nd); St, stomach

Of these, 72 were enrolled prospectively and 83 were enrolled retrospectively. Data on each rabbit's medical history, clinical examination, radiographs, blood examination, treatment and follow-up were collected. The breed of the rabbits was categorised by weight (dwarf breed <2 kg, small breed 2.1–3.75 kg, medium-sized breed 3.85.5 kg, giant breed >5.5 kg).

Inclusion criteria

Rabbits with radiographs of the abdomen or the whole body in right-sided lateral projection with a dilated stomach filled with fluid and varying amounts of gas (Figure 1) were included. Animals that died spontaneously or were euthanased within 48 hours after presentation were evaluated for criteria affecting the outcome.

Treatment at admission

Animals in shock received up to two intravenous (i.v.) boluses of 15 mL of isotonic crystalloids (Sterofundin, B.Braun Melsungen) in 20 minutes. After stabilisation, in patients without shock, fluids were given depending on their hydration status. Every patient was given at least 50 mL/kg/day maintenance as well as 5% deficit i.v. or subcutaneously (s.c.). Each rabbit received 65 mg/kg metamizole s.c. or i.v. every 8 hours (Novaminsulfon 500 mg/mL Injektionslösung, Dechra Veterinary Products) as an analgesic agent. Furthermore, all animals were given medical treatment containing a prokinetic (0.5 mg/kg metoclopramide s.c. every 8 hours, Emeprid 5 mg/mL Injektion, Ceva Tiergesundheit). Hypothermic animals were placed on a heating pad. Patients were monitored closely (body temperature, abdominal palpation, hydration status). Euthanasia was performed in cases where the animal's condition did not improve within 24–48 hours or where it deteriorated and the owners did not consent to surgery.

Radiographic examination and analysis

For the evaluation of digital radiographs, Cura-SmartClient (version 2.4.8.0) was used. Only radiographs in lateral projection with a whole abdomen were included. Abdominal fat, gastric size and the extension and position of gas accumulation in the stomach were evaluated. The same radiographic parameters were compared to radiographs of 50 rabbits without gastrointestinal diseases.

The abdominal fat was scored in all rabbits regarding the visibility of the abdominal organs. Three categories were differentiated: (1) no detail recognition/lean, (2) good detail recognition/good nutritional status and (3) obese, strong cranial/ventral displacement of the gastrointestinal tract.

For the assessment of gastric size on radiographs, a method comparable to the vertebral heart score (VHS) was developed (vertebral stomach score [VSS]). VSS was determined on lateral radiographs (Figures 2 and 3). Gastric height (dorsoventral extension [DVE]) and gastric length (craniocaudal extension [CCE]) were measured at a 90° angle to each other. The starting point for DVE was the most ventral point of the cranial margin of the vertebral body (extremitas cranialis) of the last thoracic vertebra (LTV), while the end point was the ventral stomach wall. At the widest point of the stomach, CCE was determined. The CCE and DVE stomach dimensions were transposed onto lumbar vertebrae, starting with the extremitas cranialis of the first lumbar vertebra. The summed vertebrae were expressed in lumbar vertebral units (LVU). Caudal stomach extension (CEX) was recorded in each case. The most caudal point of the tomach wall was identified by drawing a vertical line from the lumbar vertebra.

Furthermore, the extension and position of the gas accumulation in the stomach were recorded. Extension was categorised as: (1) little (>25% of gastric dimension), (2) moderate (25%–50% of gastric dimension) or (3) extensive (>50% of gastric dimension)

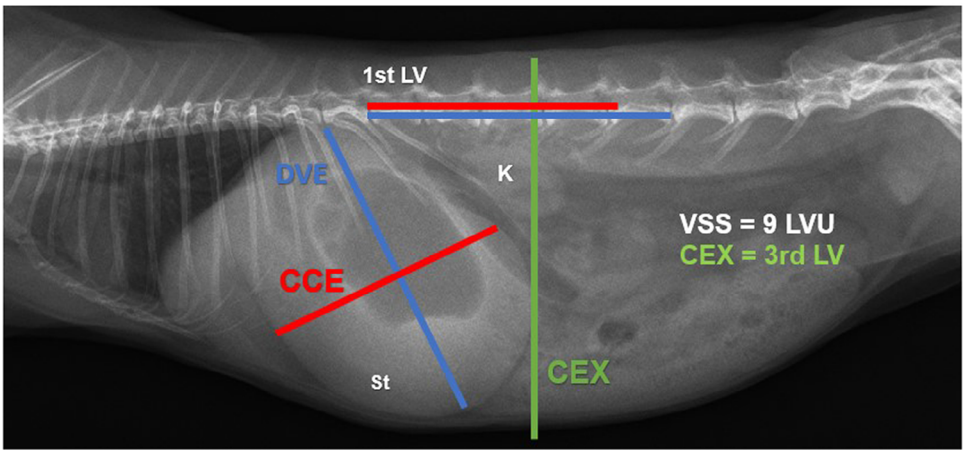

FIGURE 3 Application of the vertebral stomach score (VSS) in the right lateral radiograph of a rabbit with gastric dilation with a VSS of nine lumbar vertebrae (LV). CCE, craniocaudal extension of the stomach (red); CEX, caudal stomach extension; DVE, dorsoventral extension of the stomach (blue); K, kidney; LVU, lumbar vertebral units; St, stomach.

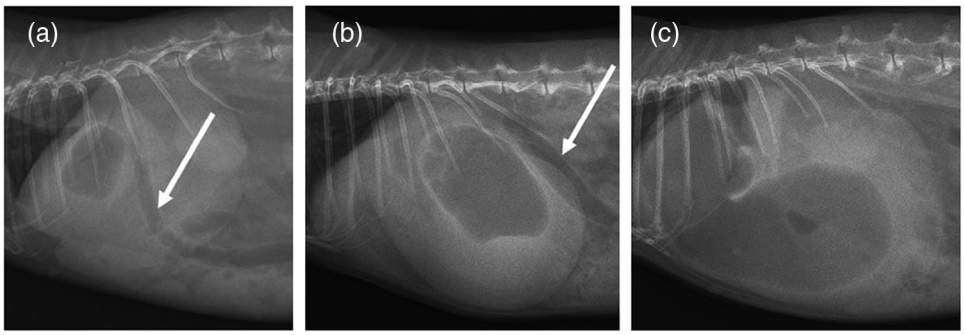

FIGURE 4 Abdominal radiographs of three rabbits showing different extensions and positions of gas accumulations in the stomach. (a) Cranial, low gas accumulation and dilated intestinal loop (white arrow), (b) central, moderate gas accumulation and dilated intestinal loop (white arrow), and (c) ventral, high gas accumulation

(Figure 4). The position of gas accumulation was classified as: (1) dorsal, (2) central, (3) ventral, (4) cranial or (5) diffuse.

Blood sampling and analysis

All blood samples were taken from the vena saphena lateralis of unsedated rabbits with a 22-G cannula (Sterican, B.Braun) after aseptic preparation. Heparin plasma was used to analyse creatinine, urea, total protein and aspartate aminotransferase (AST) with KONELAB 60I (Thermo Fisher Scientific). Potassium, sodium, free calcium and glucose were measured with GEM Premier 3000 SN 21208 (Instrumentation Laboratory). Haematology was performed from EDTA whole blood with SYSMEX XT-2000iV (Sysmex) and included haematocrit, haemoglobin, erythrocyte, platelet and white blood cell counts. In some cases, only a small amount of blood could be sampled, and only haematocrit was determined with a heparinised capillary tube (75 mm, Fa. Brand) after centrifugation (Mikrozentrifuge Heraeus Pico 17, Thermo Fisher Scientific) for 5 minutes at 13,000 g. Blood values were compared with in-clinic reference ranges obtained from 120 animals. Alterations in creatinine, glucose and sodium concentrations were classified into different grades. The classification for glucose was based on Harcourt-Brown and Harcourt-Brown:[5] hypoglycaemia (<5.7 mmol/L), normoglycaemia (5.7–15.5 mmol/L) and hyperglycaemia (>15.5 mmol/L). Hyperglycaemia was further subdivided into low (15.520 mmol/L), moderate (20.125 mmol/L) and high (>25 mmol/L). Hyponatraemia was classified as low (138–129 mmol/L) or high (>129 mmol/L), as described by Bonvehi et al.[6] Azotemia was classified, based on creatinine concentration, as low (144–300 µmol/L), moderate (300–500 µmol/L) and high (>500 µmol/L), and was differentiated into prerenal and renal according to the specific gravity of urine (prerenal: >1030; renal: 1008–1025).

Statistical analysis

Radiographic measurements for rabbits with GD were statistically compared with measurements of 50 healthy rabbits. Meanwhile, blood parameters of rabbits with GD were compared with reference values determined at our laboratory from 120 clinically healthy rabbits.

Statistical calculations were performed with SPSS (IBM Corporation, Version: 28.0.1.0 [142]). Initial testing for normality was conducted using a

Kolmogorov–Smirnov test and histograms. The significance level was set at a p-value of less than 0.05. From all parameters, median, minimum, maximum, mean and standard deviation were calculated. In order to test differences between different variables, the Mann–Whitney U-test (breed, sex, age, radiographic measurements), the Kruskal–Wallis test (blood parameters), the chi-squared test (radiographic findings, blood parameters, period of illness) and the univariate ANOVA (influence of abdominal body fat on the radiographic measurements DVE, CCE and VSS) were used. The sensitivity and specificity of the VSS and the CEX for identifying animals that did not survive to discharge were tested using receiver operating characteristic (ROC) curves. Threshold values were determined using mortality to confirm the validity of the probability of dying.

RESULTS

Signalment and clinical signs

Over a period of 6 years (2009–2015), 155 rabbits (dwarf rabbits: 54%, 84/155; small breeds: 46%, 71/155) with GD were enrolled into this study. The rabbits (25 intact males, 61 neutered males, 45 intact females, 24 neutered females) had a median age of 3 years (range: 0.25–11 years). No statistical correlation was found between the occurrence of GD and sex (p = 0.85), age (p = 0.47) or weight (p = 0.89). The majority of the patients were presented in March, May and August. Clinical signs (multiple entries) were anorexia (86%, 136/155), apathy (41%, 63/155), abdominal pain (15%, 23/155) and absent defecation (12%, 19/155). Diarrhoea was observed in 3% (5/155) of the animals. Hypothermia (body temperature below 38°C[7]) was found in 66% (102/155) of the rabbits. Rectal temperature had no impact on survival (p = 0.837).

Outcome

Of the 155 rabbits with GD, 105 survived to discharge. Four animals were euthanased at the time of admission due to poor general condition and the rejection of further treatment by the owners. All 151 of the remaining rabbits were treated conservatively at admission as there were no indications for surgery present. Of these animals, 105 survived, 31 died spontaneously and 15 were euthanased. Of these 15 rabbits, seven were euthanased without further treatment (surgical intervention declined by the owner), while eight rabbits underwent surgery 24–48 hours after admission. The surgically treated rabbits died either during (n = 1) or after surgery (n = 1) or were euthanased due to inoperable alterations such as intestinal wall strictures (n = 1), gastric wall ruptures (n = 1), a necrotic intestinal segment caused by the foreign body (n = 1) and paralytic ileus (n = 3).

TABLE 1 Radiographic measurements of the stomach in rabbits without gastrointestinal disease (lumbar vertebra units) (n = 50)

	Median	Min	Max	95th percentile	Mean	SD
DVE	3.6	2.9	4.7	2.9–4.6	3.6	0.4
CCE	3.2	2.1	5.0	2.1–4.9	3.3	0.6
VSS	6.9	5.2	9.2	5.2-9.1	6.9	1.0

Abbreviations: CCE, craniocaudal extension; DVE, dorsoventral extension; Max, maximum; Min, minimum; SD, standard deviation; VSS, vertebral stomach score.

TABLE 2 Abdominal fat scoring for vertebral stomach score in rabbits with gastric dilation (GD) (n = 149/155) and healthy control animals (n = 50)

	GD group	Control group
1	36 (24%)	10 (20%)
2	96 (64%)	28 (56%)
3	17 (12%)	12 (24%)

Causes of obstruction

In 26 animals, the causes of intestinal obstruction could be determined (by surgery: n = 8, by postmortem examination: n = 18). Trichobezoars (n = 12) in the small intestines (pylorus, duodenum, jejunum) was the main cause, followed by locus bean seeds in the jejunum (n = 5), multiple intestinal wall strictures (n = 5), absent peristalsis (paralytic ileus) (n = 3) and adhesions after ovariohysterectomy (n = 1).

Radiographic examination

Vertebral stomach score

Control group

Reference values for VSS, derived from 50 healthy rabbits with no signs of gastrointestinal disease, are presented in Table 1. The rabbits (three intact males, 16 neutered males, 24 intact females and seven neutered females) had a median age of 5 years (range: 0.25–9.5 years). The breed classification was the same as for animals with GD (dwarf rabbits: 54%, 27/50; small breeds: 44%, 20/50; medium-sized breed 2%, 1/50).

Abdominal fat (Table 2) had a significant influence on CCE (p = 0.003) and VSS (p = 0.007) in the rabbits of the control group, but not in the GD patient group ($p > 0.05$).

Gastric dilation group

VSS of rabbits with GD are presented in Table 3. The most helpful prognostic parameter in the radiographic examination was the VSS. It differed significantly between the control and GD groups (p = 0.001), with the median VSS of the GD group being 2 LVU larger than that of the control group (Tables 1 and 2 and Figure 5).

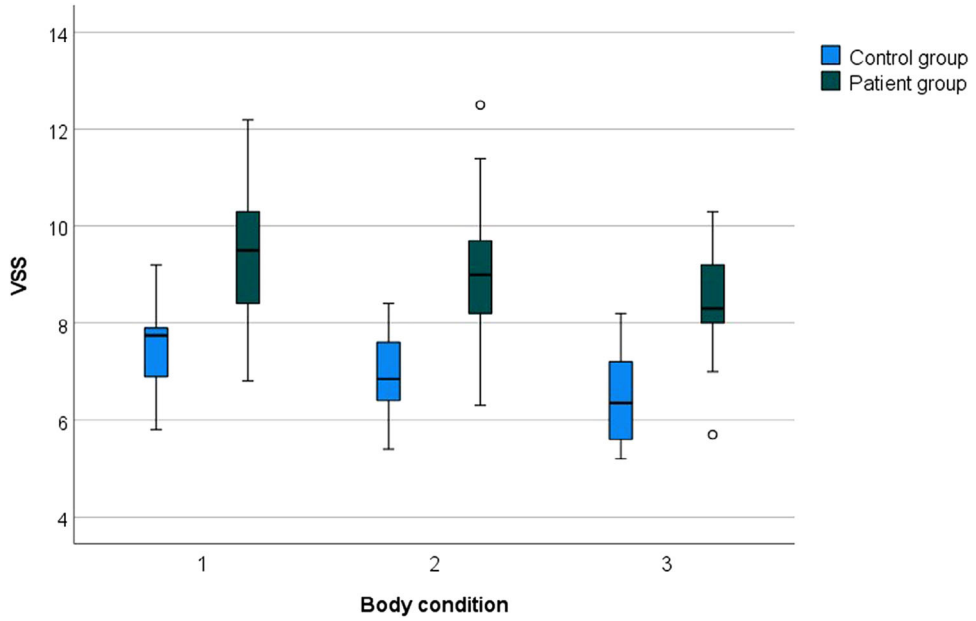

FIGURE 5 Vertebral stomach score (VSS) of the healthy controls (blue boxes) and the gastric dilation patients (green boxes) according to abdominal fat evaluated on radiographs (body condition: 1—no abdominal fat, 2—few to moderate abdominal fat, 3—high amount of abdominal fat)

TABLE 3 Radiographic measurements of the stomach in rabbits with gastric dilation (lumbar vertebra units) ($n = 155$)

	Median	Min	Max	Mean	SD
DVE	4.4	3.0	6.7	4.5	0.7
CCE	4.5	2.5	6.5	4.5	0.7
VSS	9.0	5.7	12.5	9.0	1.3

Abbreviations: CCE, craniocaudal extension; DVE, dorsoventral extension; Max, maximum; Min, minimum; SD, standard deviation; VSS, vertebral stomach score.

A cut-off value of 9.3 LVU (Figure 6) was determined to evaluate the ability of VSS to detect GD. The sensitivity of the VSS for predicting lower survival probability was 65.3% and the specificity was 67.6%. Risk assessment (odds ratio = 3.99) illustrated that the risk of a rabbit with GD dying was four times higher if its VSS was above 9.3 LVU than if it was under 9.3 LVU.

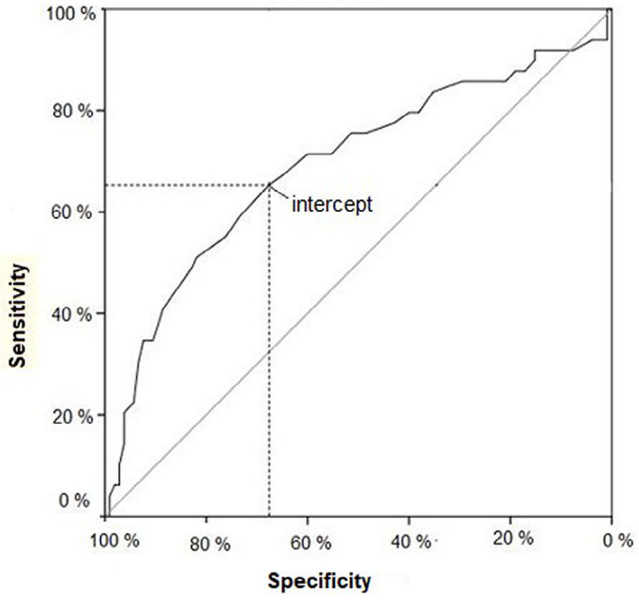

FIGURE 6 Receiver operating characteristic curve of vertebral stomach score in rabbits with gastric dilation (area under the curve= 0.692)

Caudal stomach extension

In the control group, the caudal extension of the stomach never reached the second lumbar vertebra, whereas in rabbits with GD, some stomachs expanded up to the end of the fourth lumbar vertebra (Table 4). This difference was significant ($p = 0.003$). Of the rabbits with a CEX over the second lumbar vertebra, 54% (13/24) died. In this group, mortality was twice as high as in the group of rabbits with a CEX of less than the second lumbar vertebra (27%) ($p = 0.01$).

Gastric gas quantity and distribution

Data regarding the amount and distribution of gas in the stomach are summarised in Table 5. Large amounts of gas (>50%) in the stomach ($p = 0.001$) and ventral or central location of the gas ($p = 0.023$) were associated with significantly higher mortality. In 48% (74/155) of rabbits with GD, a dilated gas-filled small intestinal loop was obvious.

Blood examination

Blood analysis was performed in 48% (75/155) of the rabbits with GD (Table 6). The most frequent biochemical changes were azotemia (51%, 37/72),

TABLE 4 Comparison of radiologically determined stomach extension in rabbits with gastric dilation (GD) and healthy controls

Caudal stomach extension	Control group ($n = 50$)	GD group ($n = 155$)
Last thoracic vertebra	17 (34%)	18 (12%)
First lumbar vertebra	26 (52%)	57 (37%)
Second lumbar vertebra	7 (14%)	56 (36%)
Third lumbar vertebra	0 (0%)	20 (13%)
Fourth lumbar vertebra	0 (0%)	4 (2%)

TABLE 5 Amount and position of gas in the stomachs of all rabbits with gastric dilation ($n = 155$) and those that did not survive to discharge ($n = 56$)

	All gastric dilation patients	Deceased animals
Amount of gas accumulation		
Little (>25%)	58 (37%)	9 (16%)
Moderate (25%–50%)	55 (36%)	15 (27%)
Extensive (>50%)	42 (27%)	24 (57%)
Position of gas		
Central[a]	56 (36%)	24 (73%)
Cranial[a]	42 (27%)	8 (19%)
Ventral[a]	31 (20%)	13 (42%)
Dorsal[a]	15 (10%)	3 (20%)
Diffuse[b]	11 (7%)	1 (9%)

[a]In order to achieve a sufficient number of samples for comparison, the positions of the gas accumulations were combined into two groups (central + ventral and dorsal + cranial).
[b]Because of small case numbers, radiographs with diffuse gas accumulation were excluded from statistical analysis.

hyperglycaemia (37%, 28/75), hyponatraemia (43%, 27/63) and increased AST activity (44%, 20/46).

Mortality for rabbits with GD and a glucose concentration above 25 mmol/L was 70% (7/10). The glucose concentration exceeded 30 mmol/L in two cases, and both of these animals died. Rabbits with azotemia (creatinine concentration above 144 µmol/L) died in 43% (16/37) of cases. Azotemia was prerenal in 20 patients ($n = 8$) and renal in eight (acute: $n = 3$; chronic: $n = 5$). In four animals, classification was not possible due to inconsistent laboratory findings. Fifty percent (4/8) of rabbits classified as prerenal were euthanased, and of those with renal azotemia, 63% (5/8) died, of which four were euthanased because of poor general condition. Postrenal azotemia did not occur. Rabbits with azotemia had significantly higher mortality than rabbits with a creatinine concentration within the reference range ($p = 0.016$). Animals with a creatinine concentration higher than 300 µmol/L had higher mortality than animals with a creatinine concentration of 144–300 µmol/L ($p = 0.001$). All rabbits with a creatinine concentration above 500 µmol/L died. Mortality in rabbits with hyponatraemia (sodium concentration below 139 mmol/L) was 36% (10/27). No statistical differences were found between the presence of hyponatraemia and survival ($p = 0.106$) and between the severity of hyponatraemia and death ($p = 0.318$).

No statistically significant differences in mortality could be observed between hypoglycaemic, normoglycaemic and hyperglycaemic rabbits ($p = 0.75$). However, by subdividing the hyperglycaemic group (low = 15.520 mmol/L, moderate = 20.125 mmol/L, high >25 mmol/L), a statistically significant difference in mortality was found between animals with a glucose concentration above 25 mmol/L and those in the other two hyperglycaemic groups ($p = 0.013$).

Animals with increased AST activity had the same outcome as rabbits without increased AST activity ($p = 0.361$).

In 20% (13/65) of the GD patients, haematocrit was increased. Eight of these animals had also azotemia (six died). Leukocytosis (leukocyte count above 14.7 G/L) was found in five rabbits and leukopenia (leukocyte count below 3.3 G/L) in three rabbits. All rabbits with leukopenia died.

DISCUSSION

GD is a common radiological finding in pet rabbits, but only a few studies have investigated this condition so far. Anorexia, apathy, abdominal pain and absent defecation are common clinical signs,[1,2,4] and these were also the main clinical findings in our study. The frequency of presentation increased in spring and summer in our population, as previously described by Brezina et al.[4] One possible cause for this seasonal presentation could be the shedding of the rabbits' winter coats during this time. Whereas all of the rabbits in the study by Brezina et al.[4] had a decreased body temperature, only one-third of the patients of our study were hypothermic. Contrary to the results of Di Girolamo et al.,[8] who had shown that rabbits with hypothermia in the course of different diseases had a higher risk of death, body temperature was not a prognostic factor for rabbits with GD in our study.

GD can be reliably diagnosed on the basis of radiographs, with typical signs being an enlarged stomach filled with fluid and gas. Harcourt-Brown,[1] as well as Schuhmann and Cope,[2] also mentioned gas-filled intestinal loops and claimed that different locations of gas were indicative for different obstruction sides (proximal and distal). Nevertheless, these classifications were based solely on radiographs without confirmation by surgery or pathology. In many of our examined patients, gas-filled intestinal loops could also be observed. However, since most of the patients were treated conservatively, this radiological finding was not further analysed. Debenham et al.[3] measured the height and width of the stomach in rabbits with GD and compared the sum of these measurements with the length of the first lumbar vertebra to the coxofemoral joint. In 98% of the examined cases of rabbits with intestinal obstruction, the value extended beyond the coxofemoral joint, whereas in the control group, only 12% exceeded this point. However, data regarding the outcome of the rabbits are lacking, and the nutritional status of the animals was not considered. While the inclusion criteria in our study were radiographs with typical signs of GD, Debenham et al.[3]

TABLE 6 Results of blood examinations in rabbits with gastric dilation

Blood parameter	*n*	Median	Min	Max	Mean	SD
Sodium (mmol/L)	63	139	115	155	138	8.3
Potassium (mmol/L)	63	4.1	2.6	12.5	4.4	1.4
Free calcium (mmol/L)	56	1.55	1.00	2.00	1.54	0.25
Glucose (mmol/L)	75	11.8	2.0	36.6	13.9	8.2
Total protein (g/L)	69	68.8	42.0	97.0	68.5	10.8
Creatinine (µmol/L)	72	148	44	583	190	125.2
Urea (mmol/L)	35	13.0	3.0	37.0	14.8	8.8
Aspartate aminotransferase (U/L)	46	53.5	10.0	3260.0	169.4	482.2
Haematocrit (L/L)	65	0.40	0.31	0.57	0.41	0.06
Haemoglobin (mmol/L)	57	8.5	6.1	11.9	8.6	1.4
Erythrocytes (T/L)	57	6.5	5.0	9.4	6.8	1.2
White blood cell (G/L)	57	9.8	2.7	24.5	10.0	4.0
Platelets (G/L)	57	417	8	1053	421	166

Abbreviations: Max, maximum; Min, minimum; SD, standard deviation.

selected only cases with surgically confirmed small intestinal obstruction. Our measurements of the stomach extension (VSS) were based on the VHS.[9] VSS proved to be a good prognostic radiological parameter for the survival of rabbits with GD. As in the study of Debenham et al.,[3] extension of the stomach beyond the second lumbar vertebra was a cutoff point. Only in rabbits with GD did the caudal gastric margin pass over the second lumbar vertebra. However, in contrast to the study of Debenham et al.,[3] where the stomach reached over the second lumbar spine in 65% of cases, we noticed this finding in only 16% of the GD cases. One possible explanation might be the different inclusion criteria of the two studies resulting in populations of animals at different disease stages.

Rabbits with extensive gas accumulations in the stomach had the highest mortality in our study, probably due to advanced disease stage causing massive gastric distention. In dogs, it is known that increased pressure in the stomach causes impaired blood flow in the gastric vessels, which in turn can lead to gastric wall oedema, thrombosis, ulceration and ultimately gastric wall rupture.[10]

Only a few publications have studied blood parameters in rabbits with GD.[4,5,11] Brezina et al.[4] diagnosed acidosis in 57% of diseased animals. In the present study, azotemia (51%), hyponatraemia (46%), hyperglycaemia (37%) and increased haematocrit (20%) were the most common abnormal blood findings in rabbits with GD. Forty-one percent of the patients with azotemia died. Due to the inability to collect urine from 72% (52/72) of the patients with azotemia, we were unable to accurately classify azotemia in these animals. Several additional parameters (haematocrit, level of creatinine and protein concentration, clinical hydration status, course of blood parameters, etc.) led to the conclusion that the majority of patients had prerenal azotemia (dehydration).

Hyponatraemia is a serious electrolyte imbalance, and 50% of the rabbits that died in our study suffered severe hyponatraemia (<129 mmol/L). Possible causes of hyponatraemia in small animals are dehydration, renal disease, shock, excessive salivation or hyperglycaemia.[12] Dehydration and shock were probably the most common causes in our rabbits. Similarly, Bonvehi et al.[6] reported 70% mortality in rabbits with undefined diseases and severe hyponatraemia.

Similar to the study of Harcourt-Brown and Harcourt-Brown,[5] our rabbits with GD frequently showed hyperglycaemia. The median glucose concentration was 21.84 mmol/L (range: 16.1–36.6 mmol/L), which is in agreement with published glucose concentrations for rabbits with intestinal obstruction (median 21.67 mmol/L).5 Hyperglycaemia in rabbits with GD is thought, among other things, to be caused by severe pain[5] and was associated with poor prognosis in horses with abdominal disease.[13] It has been suggested that the interaction of the release of glucagon, epinephrine, cortisol, growth hormones and cytokines, as well as insulin resistance, is leading to hyperglycaemia.[13] Other causes of hyperglycaemia can be obstructed urinary tract, enterotoxaemia, sepsis, acute lipolysis due to inappetence and, in very rare cases, diabetes mellitus,[5] but these conditions were not likely to be the causes of hyperglycaemia in our rabbits. Harcourt-Brown and Harcourt-Brown[5] found a very poor prognosis for patients with hyperglycaemia above 20 mmol/L, but no statistical differences in survival were found between the normoglycaemic, hypoglycaemic and hyperglycaemic rabbits with GD. Similarly, in our study, 70% (7/10) of animals with a glucose concentration higher than 25 mmol/L died, and we also did not find a relationship between the different glucose groups.

Few haematological alterations were seen in rabbits with GD, mainly an increased haematocrit (>0.46 L/L) was observed in 20% of rabbits. This probably resulted from the dehydration of the animals and is in accordance with studies from small animals suffering intestinal foreign bodies as well as in horses with colic.[14–16] Except for the above-mentioned studies, no further data on haematological and serum

biochemical parameters in rabbits with GD have been published to date. Other blood parameters that might be helpful to assess prognosis in rabbits with GD, such as lactate, have yet to be investigated.

CONCLUSION

Our results indicate that the amounts and location of gas in the stomach, as well as blood parameters such as glucose, sodium, and creatinine, may be useful prognostic parameters for rabbits with gastric dilatation.

AUTHOR CONTRIBUTIONS

Design of the study, evaluation of the data and writing of the manuscript: Anja Böttcher. *Design of the study, supervision of the findings and writing of the manuscript*: Kerstin Müller.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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DATA AVAILABILITY STATEMENT

Data are available from the authors upon request.

ETHICS STATEMENT

This study retrospectively evaluated radiographs and laboratory results obtained during routine clinical care; therefore, ethical approval was not required.

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