

Spontaneous abdominal effusion in dogs with presumed anaphylaxis

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Abstract

Objective: To describe patient characteristics of dogs developing spontaneous abdominal effusion in association with anaphylaxis.

Design: Retrospective study between 2010 and 2018.

Setting: University teaching hospital and referral specialty private practice emergency departments.

Animals: Sixteen dogs presenting to the emergency department for clinical signs suggestive of anaphylaxis with peritoneal effusion documented via ultrasonography.

Interventions: None.

Measurements and Main Results: Dogs presented with clinical signs of anaphylaxis including vomiting 12 of 16 (75%), diarrhea 9 of 16 (56%), and collapse 7 of 16 (44%). Physical examination findings included altered mentation 14 of 16 (88%), erythema 2 of 16 (13%), and urticaria 2 of 16 (13%). Historically, 3 of 16 (19%) had just recently been vaccinated, 1 of 16 (6%) had known contact with an insect, and 8 of 16 (50%) had been outdoors prior to the onset of clinical signs, whereas 5 of 16 (31%) had no known triggering event. At presentation, median systolic arterial pressure was 70 mm Hg (range, 30–210 mm Hg). Venous blood gas revealed an acidemia with a mean pH of 7.24 ± 0.09 , a base deficit of 7.15 ± 7.7 mmol/L, and an increased plasma lactate with a mean of 7.35 ± 2.09 mmol/L. Bloodwork abnormalities included an increased alanine aminotransferase activity with a mean of 439.2 ± 404.5 U/L and an increased gamma-glutamyl transferase activity with a mean concentration of 7.29 ± 4.1 U/L. Twelve patients (75%) had gallbladder wall abnormalities on ultrasonography. Eight patients (50%) had abdominocentesis performed. The median PCV of the effusion was 0.29 L/L (29%; range, 4–41) and total plasma protein was 38 g/L (3.8 g/dL; range, 2.4–6.5). Two dogs (12.5%) were euthanized. The remaining 14 dogs (87.5%) survived to discharge, with a mean hospitalization time of 23.7 ± 16.7 hours.

Conclusions: Abdominal effusion should be considered a potential sequela of anaphylaxis in dogs. Therefore, serial monitoring for presence or development of spontaneous abdominal effusion should be considered.

Abbreviations: ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; AUS, abdominal ultrasound; FAST, focused assessment with sonography for trauma; PT, prothrombin time; TPP, total plasma protein

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KEYWORDS

monitoring, resuscitation, shock, ultrasonography

1 | INTRODUCTION

Anaphylaxis is a severe allergic reaction that is rapidly progressive and can be fatal.¹ It is a systemic type I hypersensitivity reaction with potential involvement of multiple organ systems.² Drugs (antimicrobials, vaccines, nonsteroidal anti-inflammatory drugs, opiates), foods, insect bites, snake venom, and blood products are among the most common causes of immunological, immunoglobulin E-mediated and non-immunoglobulin E-mediated anaphylactic reactions cited in the veterinary literature.³ Cold, exercise, water, and heat have also been noted as nonimmunological causes of anaphylaxis in people.⁴ Clinical signs can widely vary, but the most commonly affected organ systems are the cutaneous, respiratory, cardiovascular, and gastrointestinal systems. As “shock organs” differ by species, clinical signs may be varied and species dependent. In dogs, the major organ systems affected are the gastrointestinal tract and the liver. This typically manifests as vomiting, diarrhea, hemorrhagic enteritis, and hepatic venous congestion, causing portal hypertension and visceral pooling of blood.⁵ This can potentially quickly progress to cardiovascular compromise, collapse, seizure activity, coma, and subsequent death. Regardless of the etiologic agent and manifestation of the reaction, epinephrine administration and supportive care remain the treatment of choice as described in recent anaphylaxis guidelines.⁶

There are very few published reports of anaphylactic reactions in the veterinary literature; consequently, much of the immunological and physiological data are based on human literature. Recent case reports in dogs have revealed significant antemortem changes to the liver and the gallbladder, with portal vein hypertension, sinusoidal congestion, and increases in alanine aminotransferase (ALT) activity, as well as a thickened and striated gallbladder wall.^{7–9} Spontaneous abdominal effusion in dogs with anaphylaxis has been noted previously but has not been described in detail.^{9–11} The objective of this retrospective study is to describe the presenting characteristics of dogs with documented spontaneous abdominal effusion after presumptive diagnosis of anaphylaxis.

2 | MATERIALS AND METHODS

2.1 | Study population and clinical evaluation

The electronic medical record systems of contributing institutions were searched for patients with “anaphylaxis” included in their medical record between January 2010 and May 2018. These records were then examined to determine which had a clinical picture consistent with anaphylaxis and which of these had either a focused assessment with sonography for trauma (FAST scan) or full abdominal ultrasound (AUS) performed at any point in their hospitalization. It was then fur-

ther determined which had documentation of ultrasonographic evidence of peritoneal effusion. The corresponding medical records for these patients were then reviewed. Inclusion criteria were as follows: dogs with acute onset of clinical signs consistent with anaphylaxis (eg, vomiting, diarrhea, urticaria, erythema, or altered mentation) that were temporally associated with a known potential trigger, such as vaccines or exposure to an insect, or could not be readily explained by another cause of documented hypotension (systolic blood pressure ≤ 100 mm Hg or presumed hypotension based on clinical signs of collapse or obtunded/stuporous mentation); and documented ultrasonographic evidence of peritoneal effusion during the hospitalization period. Exclusion criteria were as follows: noncanine patients; patients without indication of anaphylaxis (such as patients in which anaphylaxis was mentioned as a possible sequela of their diagnosis of mast cell disease or snake bite envenomation); patients with peritoneal effusion that could be explained by another condition (such as observed neoplasia on imaging); patients that were ultimately diagnosed with, or were known to have, a concurrent disease that could explain any of the previously mentioned abnormalities; and insufficient medical records to adequately characterize the patient as having likely experienced anaphylaxis (Figure 1).

Data retrieved from the medical records included signalment, pertinent medical history, clinical signs, recent exposure to a potential allergen (eg, vaccines, insects, recently outside), as well as body weight (kg), vital signs (temperature, heart rate, respiratory rate, mucus mem-

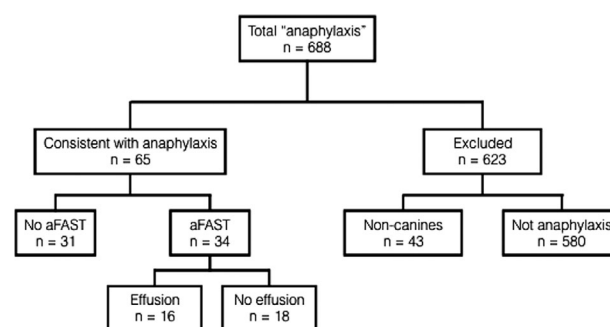


FIGURE 1 Electronic medical records were searched for the term “anaphylaxis” and these charts were reviewed

Note: Patients were excluded if they were non-canine or did not have clinical signs consistent with anaphylaxis. Patients were considered as having suspected anaphylaxis if they had acute onset of vomiting, diarrhea, urticaria, erythema, collapse, or altered mentation as well as documented hypotension (systolic blood pressure < 100 mm Hg) or suspected hypotension based on clinical signs of collapse or altered mentation, with none of these signs being readily explained by another cause. These charts were then further examined to see if abdominal FAST or abdominal ultrasound was performed and, if so, whether peritoneal effusion was documented. Cases with peritoneal effusion were included in this study

TABLE 1 Patient population

Data	Presenting characteristics
Age (y)	4.5 (0.83–14)
Weight (kg)	10.55 (4.65–55.4)
Historical information	
Vaccination prior to presentation (%)	3/16 (18.8)
Known contact with an insect (%)	1/16 (6.2)
Outdoors prior to onset of clinical signs (%)	8/16 (50)
No known triggering event (%)	5/16 (31)
Clinical signs	
Vomiting (%)	12/16 (75)
Diarrhea (%)	9/16 (56)
Collapse (%)	7/16 (43.8)
Physical examination findings	
Altered mentation (%)	14/16 (87.5)
Erythema (%)	2/16 (12.5)
Urticaria (%)	2/16 (12.5)
Admission parameters	
Temperature (°C/°F)	37.7 ± 2.0/99.8 ± 2.0
Heart rate (/min)	160 ± 41
Respiratory rate (/min)	34 ± 13

Note: Patient data including age, weight, clinical signs, physical examination findings, and admission parameters. Normally distributed data are presented with mean and standard deviation, and nonnormally distributed data are presented as median and range.

brane color, capillary refill time), and significant physical exam findings (such as mentation, presence of urticaria or erythema), any blood work (CBC,^{*,†} serum biochemical profile,^{‡,§} venous blood gas,^{**} prothrombin time [PT] and activated partial thromboplastin time [aPTT],^{††} PCV, total plasma protein [TPP]) performed, and ultrasonographic assessment of peritoneal effusion at the time of presentation and during serial rechecks when applicable. When available, PCV and TPP of peritoneal effusion obtained by abdominocentesis were also recorded. Given the retrospective nature of the study, long-term patient follow-up was not consistently available for this patient population and thus was not evaluated.

2.2 | Statistical analysis

Continuous numerical data were assessed for normality using the D'Agostino and Pearson normality. Normally distributed data are presented as mean ± standard deviation; nonnormally distributed data are presented as median (range).

3 | RESULTS

3.1 | Patient population

A total of 688 records included the term “anaphylaxis.” Of these, 623 were excluded as non-canine patients ($n = 43$) or not having signs

consistent with anaphylaxis ($n = 580$). A total of 65 dogs had clinical signs consistent with anaphylaxis, with 31 (47.7%) excluded as no documented abdominal FAST or AUS was performed. Of the 34 (52%) dogs with abdominal FAST or AUS, 16 dogs had peritoneal effusion and satisfied the inclusion criteria. These included 10 (62.5%) neutered females, 5 (31.2%) neutered males, and 1 (6%) intact female. The median age was 4.5 years (range, 0.83–14 y). Breeds represented were mixed breed ($n = 5$), Staffordshire Terrier ($n = 2$), and 1 each of the following breeds: Cairn Terrier, Pug, Dachshund, Yorkshire Terrier, Chihuahua, Pit Bull Terrier, Rottweiler, Cavalier King Charles Spaniel, and Siberian Husky. Patient data including age, weight, clinical signs, physical examination findings, and admission parameters are presented in Table 1.

3.2 | Diagnostics

Fifteen patients had an abdominal FAST scan performed at presentation. Subjective assessment of the fluid volume was noted in the medical record and revealed the following volumes of effusion: none ($n = 3$), scant ($n = 6$), mild ($n = 2$), moderate ($n = 3$), and severe ($n = 1$). Of the 3 dogs with no effusion documented at presentation, 2 had moderate effusion and 1 had mild effusion documented within 2–3 hours of fluid resuscitation. Figure 2 reveals a patient with effusion that was documented to be hemorrhagic in nature. Ultrasound was not performed at the time of presentation in 1 patient, but this

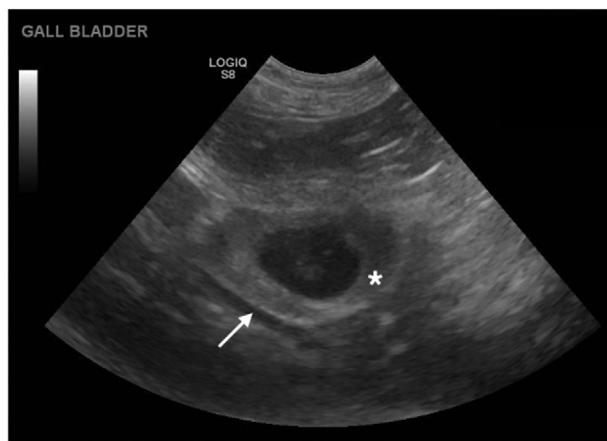


FIGURE 2 Ultrasound image from one of the patients showing gallbladder wall edema (*) and peritoneal effusion (arrow) that was confirmed to be hemorrhage on abdominocentesis

patient was documented to have moderate effusion on AUS performed 13 hours later. Eight patients had aspiration of the fluid, with 7 patients having a recorded PCV (median, 0.29 L/L [29%]; range, 4–41%) and TPP (median, 38 g/L [3.8 g/dL]; range, 24.0–65.0 g/L [2.4–6.5 g/dL]). The remaining patient had a grossly hemorrhagic effusion, although a PCV/TPP was not recorded.

Imaging (via FAST scan or full AUS) revealed gallbladder abnormalities including a subjectively thickened gallbladder wall ($n = 6$) and gallbladder wall edema ($n = 6$). Figure 2 displays an ultrasound image of a patient with gallbladder wall edema and peritoneal effusion. Four patients were not appreciated to have gallbladder abnormalities on imaging. Arrhythmias were noted in 6 (37.5%) patients, with some patients having more than 1 type of arrhythmia. Four patients (25%) were noted to have ventricular premature complexes, with 1 of these patients progressing to ventricular tachycardia. One patient (6.25%) had sinus bradycardia, whereas 2 patients (12.5%) had sinus tachycardia. One patient (6.25%) also had occasional atrial premature complexes. A baseline cortisol was performed on 2 patients, with values reported as 3.5 $\mu\text{g/dL}$ and $>10 \mu\text{g/dL}$, indicating that neither were classified as having hypoadrenocorticism.

Available results from blood gas values, CBC, and serum biochemical profile are presented in Table 2. The median systolic arterial pressure at presentation was 70 mm Hg (range, 30–210 mm Hg). The initial PCV at presentation for patients was a mean of 0.55 ± 0.13 L/L ($55 \pm 13\%$), with a TPP of 55 ± 14 g/L (5.5 ± 1.4 g/dL). Repeat PCV/TPP was performed 9.7 $\pm$ 4.3 hours after presentation. The median difference between the peripheral PCV and the respective abdominal effusion PCV was 0.18 L/L (18%), with a range of 0.08–0.62 L/L (8–62%). The repeat peripheral PCV/TPP for these patients was 0.42 ± 0.11 L/L ($42 \pm 11\%$) and 50 ± 11 g/L (5.0 ± 1.1 g/dL), respectively. Blood gas analysis revealed that these patients had an acidemia characterized by a metabolic acidosis and hyperlactatemia, as well as ionized hypocalcemia and hyperglycemia (Table 2). Significant findings on serum biochemical profile included moderate increase in ALT and mild increase in gamma-glutamyl transferase activities. The remainder of

the serum biochemical profile, as well as the CBC, was largely unremarkable for these patients. A PT and aPTT were performed in 5 of the 16 patients, with only 1 patient having a PT that was prolonged greater than 25% above the reference interval (PT = 38 s; reference interval, 14–19 s) and 1 patient having an aPTT that was significantly prolonged (PTT > 200 s; reference interval, 75–100 s).

3.3 | Treatments

On presentation, 12 dogs received diphenhydramine, 10 received dexamethasone, 5 received epinephrine, 3 received ondansetron, and 2 received maropitant. Twelve dogs received crystalloid fluid bolus(es) at presentation, with a mean total administration of 41.1 ± 22.6 mL/kg during fluid resuscitation. One dog received a packed red blood cell transfusion, and 1 dog received a fresh frozen plasma transfusion. After initial fluid resuscitation, these patients received crystalloid fluid therapy, with a mean dose of 4.15 ± 2.15 mL/kg/h. The mean duration of fluid therapy administration was 19.2 ± 15.3 hours.

3.4 | Outcome

Fourteen patients (87.5%) survived to discharge. Mean hospitalization time for surviving patients was 27.1 ± 15.7 hours. Two (12.5%) patients were euthanized within hours of presentation due to the severity of their signs and the anticipated financial burden of continued care. Necropsy was not performed on these cases. Follow-up was not available on the surviving patients.

4 | DISCUSSION

In this study, we described the presenting characteristics of 16 dogs that developed spontaneous peritoneal effusion secondary to anaphylaxis, which was found to be consistent with hemorrhage in all of the patients that had abdominocentesis performed. There is a dearth of information in veterinary medicine documenting the occurrence of abdominal effusion, and specifically hemoperitoneum, as a sequela of anaphylaxis.^{9–11} In this retrospective observational study, we found that the majority of these patients displayed an altered mentation at presentation and were tachycardic, hypothermic, and hypotensive. These patients had recently been outdoors or vaccinated and showed gastrointestinal clinical signs, including vomiting and diarrhea. Blood gas analysis revealed metabolic acidosis with an associated hyperlactatemia, changes consistent with cardiovascular compromise. Serum biochemical profile abnormalities revealed increases in ALT and gamma-glutamyl transferase activities. Ultrasonographic findings were consistent with those previously reported to be associated with anaphylaxis, including gallbladder wall edema and wall thickening.¹⁰ The majority of these patients survived to discharge (87.5%), and 2 were euthanized shortly after presentation due to the financial burden associated with continued hospitalization and supportive care.

TABLE 2 Diagnostics

Data	Presenting values	Reference interval	Data	Presenting values	Reference interval
Venous blood gas			CBC		
pH	7.24 ± 0.09	7.35–7.47	Hematocrit (L/L) [%]	0.54 ± 0.11 [37.3–61.7]	0.37–0.62 [37.3–61.7]
pCO ₂ (mm Hg)	42.8 ± 13.91	32–43	Hemoglobin (mmol/L) [g/dL]	12.02 ± 2.28 [19.37 ± 3.68]	8.13–12.72 [13.1–20.5]
HCO ₃ (mmol/L)	17.78 ± 4.13	20–25	RBC (mL/μL)	7.74 ± 1.63	5.65–8.87
Hematocrit (L/L) [%]	0.51 ± 0.10 [51.36 ± 10.48]	–	MCV (fL)	69.86 ± 3.13	61.6–73.5
Total Hb (mmol/L) [g/dL]	10.47 ± 2.02 [16.87 ± 3.26]	4.96 ± 9.31 [8–15]	MCH (pg)	24.5 (23.4–30.3)	21.2–25.9
Sodium (mmol/L)	144.7 ± 4.20	140–150	MCHC (g/dL)	36.06 ± 2.23	32.0–37.9
Potassium (mmol/L)	3.80 ± 0.42	3.9–4.9	Leukocytes (x10 ⁹ /L)	10.74 ± 5.67	5.05–16.76
Chloride (mmol/L)	115.8 ± 3.62	109–120	Neutrophils (x10 ⁹ /L)	10.24 (1.48–75.3)	2.95–11.64
Calcium (mmol/L) [mEq/L]	1.74 ± 0.94 [3.47 ± 1.88]	2.5–3.0 [5.0–6.0]	Lymphocytes (x10 ⁹ /L)	2.11 (0.41–23)	1.05–5.1
Magnesium (mmol/L) [mEq/L]	0.62 (0.53–1.3) [1.24 (1.06–2.6)]	0.52–0.73 [1.04–1.46]	Monocytes (x10 ⁹ /L)	1.23 ± 1.43	0.16–1.12
Glucose (mmol/L) [mg/dL]	7.2 ± 2.59 [129.8 ± 46.72]	4.72–6.22 [85–112]	Eosinophils (x10 ⁹ /L)	0.25 (0.04–4.2)	0.06–1.23
Plasma lactate (mmol/L)	7.35 ± 2.09	0–2	Basophils (x10 ⁹ /L)	0.04 ± 0.07	0–0.1
BUN (mmol/L) [mg/dL]	6.16 ± 1.66 [17.26 ± 4.66]	3.21–11.78 [9.0–33]	Platelet count (x10 ⁹ /L)	163.7 ± 53.51	148–484
Creatinine (μmol/L) [mg/dL]	85.77 ± 27.41 [0.97 ± 0.31]	61.89–159.16 [0.7–1.8]			
Base deficit (mmol/L)	7.15 ± 7.67				
Serum Biochemical Profile					
Albumin (g/L) [g/dL]	30 ± 4.3 [3.0 ± 0.43]	29–42[2.9–4.2]	Total bilirubin (μmol/L) [mg/dL]	6.84 ± 6.84 [0.4 ± 0.4]	1.71–6.84 [0.1–0.4]
Globulin (g/L) [g/dL]	22.9 ± 6.7 [2.29 ± 0.67]	22–29[2.2–2.9]	Cholesterol (mmol/L) [mg/dL]	4.06 ± 1.23 [157.1 ± 47.4]	2.07–8.15 [80–315]
ALT (μkat/L) [U/L]	7.29 ± 6.71 [439.2 ± 404.5]	0.17–0.91 [10–55]	Calcium (mmol/L) [mg/dL]	2.34 ± 0.2 [9.37 ± 0.81]	2.32–2.89 [9.3–11.6]
ALP (μkat/L) [U/L]	1.18 (0.47–3.75) [71 (28–225)]	0.25–2[15–120]	Phosphate (mmol/L) [mg/dL]	1.64 ± 0.57 [5.09 ± 1.78]	1.03–2.62 [3.2–8.1]
GGT (μkat/L) [U/L]	0.12 ± 0.07 [7.29 ± 4.11]	<0.05–0.1 <3–6			

Note: Variables such as venous blood gas values, CBC, and serum biochemical profile are displayed. Normally distributed data are presented with mean and standard deviation, and nonnormally distributed data are presented as median and range. Reference intervals for venous blood gas, CBC, and serum biochemistry are shown.

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume.

Dogs with anaphylaxis can present with a wide variety of clinical signs that vary in severity, and diagnosis is based primarily on a thorough history and recognition of clinical signs.¹⁰ Often there is a sudden onset of clinical signs within minutes to hours of exposure to an allergen with potential for rapid progression.⁶ Patients can present with urticaria, pruritus, restlessness, vomiting, diarrhea, upper airway signs (with or without obstruction), tachycardia, pale mucous membranes, seizures, coma, and hypotensive shock.³ The World Allergy Organization has published guidelines in the human literature for diagnostic cri-

teria of anaphylaxis.⁶ For anaphylaxis to be considered highly likely, 1 of 3 different scenarios may be present. In the first scenario, there is a sudden onset of clinical signs (eg. involvement of the skin, mucous membranes, itching) within minutes to hours, and potentially respiratory symptoms, a reduction in blood pressure, or development of organ dysfunction. In the second scenario, there is a known exposure to a likely allergen, as well as at least 2 of the following symptoms: sudden skin or mucosal signs, sudden respiratory signs, sudden reduced blood pressure, or sudden gastrointestinal symptoms. In the third diagnostic



scenario, there is exposure to a known allergen as well as a decrease in systolic blood pressure. These guidelines were used to support the diagnostic criteria for anaphylaxis in this study.

Dogs with anaphylaxis can experience significant venous congestion. Sonographically, this is represented by distension of the caudal vena cava as well as the hepatic veins appearing as tree trunks ("tree trunk sign") and gallbladder wall edema ("gallbladder halo sign").^{7,10} The gallbladder wall may appear thickened and striated with alternating echogenicity.¹⁰ Alternatively, the caudal vena cava may appear flattened due to lack of venous return. It is important to note that the gallbladder halo sign is not pathognomonic. There are a number of other important differential diagnoses, including sepsis, pericardial effusion, cardiac tamponade, third spacing, and right-sided heart failure.^{7,10,12} Additionally, gallbladder wall edema is an immediate finding in dogs with anaphylaxis, as opposed to an increased ALT activity, which may take 2–4 hours to increase.⁷ Therefore, a finding of gallbladder wall edema, with concurrent history or clinical signs, may be an indication that a patient is experiencing anaphylaxis. These changes were also used to aid in diagnosis of anaphylaxis in our patients.

As described herein, dogs with anaphylaxis may also present with spontaneous peritoneal effusion, specifically hemoperitoneum. The pathophysiology of hemoperitoneum with anaphylaxis is unclear, and reports are scarce both in human and veterinary literature. Case reports exist in the human literature identifying disseminated intravascular coagulation as a complication of anaphylaxis.^{13–16} Although coagulopathies and disseminated intravascular coagulation are potential causes of hemoperitoneum, it is unlikely to explain hemoperitoneum in the cases described in the current report, as all platelet counts were normal and only 1 patient had a prolonged PT and 1 patient had a prolonged aPTT. It should be noted, however, that PT/aPTT were only assessed in 5 patients. In a retrospective analysis of 60 cases of non-traumatic hemoperitoneum in dogs, the spleen was thought to be the primary source of bleeding, with the liver being the second most common source.¹⁷ However, 68% of these cases were neoplastic, which did not appear to be a cause in our patients. It is possible in anaphylaxis that rapid splenic or liver engorgement and subsequent vascular rupture of 1 or both of these organs could be the cause of the hemoperitoneum. It is known that dogs in anaphylaxis develop marked hepatic venous congestion and increased vascular permeability.³ Moreover, increased hepatic venous pressure is known to cause sinusoidal congestion and exudation of protein and fluid into the space of Disse, which contains blood plasma.¹⁸ In these patients, it is possible that widespread mast cell degranulation with release of heparin and tryptase, along with significant hepatic venous congestion, could contribute to hemoperitoneum due to diapedesis of blood and plasma, despite relatively unremarkable changes in coagulation profiles.^{10,19}

Given that peritoneal effusion can be a potential sequela of anaphylaxis, coagulation profiles and serial FAST scans should be considered in all patients with suspected anaphylaxis.^{10,20} cursory bedside AUS scans can be used, not only to detect free fluid in the abdomen but also for detection of the "gallbladder halo" sign. Importantly, clinicians should always consider the possibility of abdominal effusion

when a patient presents with anaphylaxis, especially if there is a failure to respond to treatment as expected or if a recurrence of shock signs is seen after initial stabilization. Moreover, short-term hospitalization with monitoring should be considered, as abdominal effusion can develop post-stabilization.

Direct intervention for the peritoneal effusion, such as surgery or blood product administration, was not needed, suggesting the cause of hemorrhage was transient and spontaneously resolved. This would suggest that medical management, rather than surgical management, is indicated in these cases. Therefore, epinephrine administration and supportive care remain the recommended course of treatment for anaphylaxis,⁶ as was administered to the patients reported in this study. Prognosis for nontraumatic hemoperitoneum has often been considered poor, as it is generally associated with a neoplastic cause.²¹ Although the sample size was limited, the patients in this study seemed to have a good prognosis as 14 of 16 survived to discharge, with the remaining 2 of 16 patients being euthanized soon after presentation due to severity of signs and anticipated financial burden of continued care. These 2 patients were noted to have only mild effusion at presentation.

The retrospective nature of this study was an obvious limitation. The sample size was small, with only 16 patients meeting inclusion criteria in the time period examined. This may attest to the rarity of this disease process or, more likely, the fact that it is not identified when it does occur, as clinicians are unaware of this potential sequela to anaphylaxis. Additionally, the diagnosis of anaphylaxis can be difficult, relying heavily on history, physical examination, and associated clinical findings, so it is possible that some cases were missed in our medical records search. In this study, we identified patients that had medical records that would support the diagnosis of anaphylaxis, although there is no way to verify that these patients were truly experiencing anaphylaxis. Although the majority of samples obtained by abdominocentesis were consistent with hemorrhage, this was only performed in half of the patients. This may be due, in part, to an inability to obtain a sample given that there was a scant amount of effusion noted in several patients. As such, it is uncertain whether the effusion associated with anaphylaxis is always hemorrhagic in nature, as 1 of the patients reported here had a PCV of only 0.04 L/L (4%) when abdominocentesis was performed.

This study describes dogs with presumed anaphylaxis and spontaneous abdominal effusion. Prospective studies of dogs with anaphylaxis are needed to fully elucidate the incidence of spontaneous abdominal effusion in this patient population, to determine the mechanism by which this effusion occurs and the most appropriate treatment strategies. Awareness of this sequela and serial assessment for peritoneal effusion will be important for further characterization of this condition. Clinicians that suspect anaphylaxis in their patients should consider performing imaging to assess for the occurrence of abdominal effusion and assess for gallbladder wall changes that may be supportive of a diagnosis of anaphylaxis. Serial abdominal imaging should be considered as some patients develop effusion hours after fluid resuscitation.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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END NOTES

- * ElementHT5, Heska, Loveland, CO.
- † Hematology analyzer, ADVIA 2120i, Siemens, Berlin, Germany.
- ‡ ElementDC, Heska, Loveland, CO.
- § Serum biochemistry panel, COBAS c501, Roche, Basel, Switzerland.
- ** Stat Profile pHox, NOVA Biomedical, Waltham, MA.
- †† EPOC Reader, Siemens, Tarrytown, NY.
- ‡‡ Coag Dx Analyzer, IDEXX Laboratories, Inc., Westbrook, ME.

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