

RETROSPECTIVE STUDY

Anaphylaxis-related hemoperitoneum in 11 dogs

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Objective: To describe the unique complication of hemoperitoneum associated with anaphylaxis.

Design: Retrospective case series from September 2012 to August 2017.

Setting: Two private emergency and specialty referral hospitals.

Animals: Eleven client-owned dogs diagnosed with anaphylaxis and hemoperitoneum upon presentation or referral.

Interventions: None.

Measurements and Main Results: Inclusion criteria included clinical signs consistent with anaphylaxis (hypotension, tachycardia, vomiting, diarrhea, weakness, collapse, with or without the presence of dermal signs) due to witnessed or unwitnessed presumed bee sting, an elevated alanine aminotransferase (ALT), performance of abdominal FAST (AFAST) examination with an abdominal fluid score, the sonographic presence of gallbladder wall edema, and hemoperitoneum.

All dogs (n=11) were managed medically without surgical intervention. 91% (n=10) of dogs survived to discharge.

Conclusions: Hemoperitoneum development can be seen with anaphylactic reactions, though the exact mechanism remains to be fully understood. Medical therapy is warranted and can be successful in these patients; surgery is not indicated to address hemoperitoneum.

KEYWORDS

anaphylaxis, cardiovascular, coagulation, coagulopathies, clinical pathology, critical care, hemodynamics, hypotension, shock

1 | INTRODUCTION

Anaphylaxis (AX) is a sudden-onset, systemic, potentially fatal, hypersensitivity reaction.¹ The effects of anaphylactic reactions are manifest

by multiple organ systems; a spectrum of severity can be seen. The main shock organs in the dog are the hepatic and gastrointestinal systems.¹ Thus, typical clinical signs and diagnostic findings in canine AX include hemorrhagic gastroenteritis, hepatic congestion, and gallbladder wall edema, increased liver enzyme activities, and hemoconcentration, with or without cutaneous signs.² AX-related coagulopathy occurs and may manifest as nontraumatic hemoperitoneum, which has not been extensively described with AX in veterinary medicine, other than in a single abstract³ and, most recently, a case report.³ Only 1 report of AX-related hemoperitoneum has been found within the human literature.²

Abbreviations: AFAST, abdominal focused assessment with sonography for trauma; AFS, abdominal fluid score; ALT, alanine aminotransferase; AX, anaphylaxis; BG, blood glucose; BK, bradykinin; H1R, histamine 1 receptor; H2R, histamine 2 receptor; H3R, histamine 3 receptor; H4R, histamine 4 receptor; IgE, immunoglobulin E; NO, nitric oxide; PAF, platelet activating factor; PT, prothrombin time; aPTT, activated partial thromboplastin time; TFAST, thoracic focused assessment with sonography for trauma; TPP, total plasma protein; Vet BLUE, Veterinary Bedside Lung Ultrasound Examination

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Furthermore, the etiology of hemoperitoneum associated with AX has not been definitively characterized, though likely heparin, bradykinin (BK), tryptase, histamine, platelet activating factor (PAF), and other factors play a role.

The following is a retrospective case series of 11 dogs with AX-related hemoperitoneum that describes their clinical course and outcome.

2 | MATERIALS AND METHODS

2.1 | Criteria for selection of cases

Our retrospective case series enrolled dogs with AX presented to 2 emergency and specialty referral hospitals from September 2012 to August 2017. Inclusion criteria were previously healthy dogs with acute onset of clinical signs consistent with AX (hypotension, tachycardia, vomiting, diarrhea, weakness, collapse, with or without the presence of dermal signs)^{4,5} and biomarkers found to be significantly associated with AX (the sonographic presence of gallbladder wall edema and increased alanine transaminase [ALT] activity).¹ Gallbladder wall edema was defined as the presence of intramural sonographic striation, referred to as the "halo effect" or "double-rim effect."¹ In addition to meeting the criteria for AX, the patients included also had hemoperitoneum, identified upon performance of abdominal focused assessment with sonography for trauma (AFAST) or a complete abdominal ultrasound examination. Hemoperitoneum was defined as a peritoneal effusion having a PCV $\geq 25\%$ the peripheral PCV.⁶ Patients were excluded if there was known trauma within the last 24 hours, anticoagulant rodenticide toxicosis within the last month, or another cause for hemoperitoneum was identified (eg, abdominal neoplasia, septic focus).

2.2 | Medical records review

Data were recorded on commercially available spreadsheet programs^{†,‡} and included patient signalment, presenting complaint, temperature, heart rate, respiratory rate, presence or absence of cutaneous signs, witnessed sting or insect bite, PCV, and total plasma protein (TPP) of peripheral blood, blood glucose (BG), lactate, blood pressure, ALT, prothrombin time (PT), activated partial thromboplastin time (aPTT), initial AFAST with an abdominal fluid score (AFS) and recording of the AFAST-positive sites,⁷ the presence of a "halo effect,"¹ complete abdominal ultrasound findings, peritoneal effusion PCV and TPP, administration of epinephrine, other medications (histamine 1 or 2 receptor [H1R, H2R] antagonists, glucocorticoids), and transfusion products (fresh frozen plasma and packed red blood cells), thoracic focused assessment with sonography for trauma (TFAST),^{8,9} and lung ultrasound (Vet BLUE protocol)^{10,11} findings, serial AFAST with AFS, length of hospitalization, survival, and AX recurrence, if any.

All dogs had a serum biochemistry panel with electrolytes and an AFAST^{8,9} performed, with notation of gallbladder wall characteristics. Additional diagnostics included Doppler^{§,¶} or oscillometric blood

pressure (mean or systolic) assessment, PCV and TPP, BG,^{#,||} plasma lactate,^{**,**††} coagulation testing (PT^{‡‡} and aPTT^{§§}), abdominocentesis, abdominal effusion analysis (PCV and TPP), and serial AFAST during hospitalization. Complete abdominal ultrasound by a boarded specialist (radiologist, criticalist, or internist) was performed in 6 dogs. The other 6 dogs had a Global FAST (combination of AFAST, TFAST, and Vet BLUE) performed by a veterinary criticalist.¹² Hypotension was defined as systolic blood pressure < 90 mm Hg or mean arterial pressure < 60 mm Hg. Hemoconcentration was defined as PCV or Hct ≥ 0.55 L/L (55%). Hypoglycemia was defined as BG concentration < 3.33 mmol/L (60 mg/dL). Hyperlactatemia was defined as plasma lactate > 2.5 mmol/L. Coagulopathy was defined by a prolongation of PT and aPTT $> 25\%$ of the upper reference limit but not greater than a 3-fold increase in PT.

Hospitalized cases were designated as nonsurvivors if they died or were euthanized in hospital; survivors if they were discharged. Follow-up was performed by phone call to clients and referring hospitals for information regarding recurrence of clinical signs and long-term outcome.

3 | RESULTS

Twenty-five dogs met the criteria for AX with abdominal effusion identified during initial evaluation. Sample acquisition via abdominocentesis was performed in 68% of dogs ($n = 17$); 8 dogs were excluded from further analyses because abdominocentesis was not performed. Four of the 17 dogs were excluded due to lack of confirmation of hemoperitoneum. One dog had undergone cholecystectomy and was excluded due to inability to evaluate the gallbladder wall. One final dog was excluded due to a PT measurement greater than the upper reference limit with no definitive value. Thus, a total of 11 dogs satisfied all selection criteria and were included in the study. Signalment included 4 neutered females, 1 intact female, 5 neutered males, and 1 intact male. Median patient age and body weight were: 4.0 years (range, 0.83 to 9), 21.6 kg (2.73 to 54). Breeds represented included German Shepherd (1), Terrier mix (2), Basenji mix (1), Hound mix (2), Labrador mix (1), Bichon Frise mix (1), Catahoula mix (1), Pit Bull (1), and Rottweiler (1).

3.1 | Presentation and clinical signs

All dogs ($n = 11$) presented with collapse or acute weakness. Secondary complaints included vomiting ($n = 6$), diarrhea, and often hematochezia ($n = 6$). None were described to have dermal manifestations (erythema, pruritus, urticarial, angioedema). One dog was found in the proximity of a dead bee, though no sting incident was witnessed. Several dogs ($n = 4$) were on a walk or outside prior to collapse, with a few ($n = 2$) displaying acute signs of lameness (presumably due to insect bite or sting).

Median patient data and ranges for presenting vital signs were collected: body temperature 37.9°C (100.2°F) (range, 37 to 39.2°C [98.6 to 102.5°F]) ($n = 11$), heart rate 180/min (range, 98 to 200/min) ($n = 11$), and respiratory rate 40/min (range, 28 to 60/min) ($n = 11$).



3.2 | Diagnostics

Abdominocentesis and fluid analysis were used to confirm hemorrhagic effusion in the 11 included dogs. AFSs were recorded: AFS 1 ($n = 2$), AFS 2 ($n = 1$), AFS 3 ($n = 4$), AFS 4 ($n = 4$) with the frequency of positive sites: diaphragmatico-hepatic (9), spleno-renal (7), cysto-colic (8), and hepato-renal (7), respectively. Halo effect was noted in all 11 cases. Median abdominal effusion PCV was 27.5% (range, 24 to 60). Median abdominal effusion TPP was 46 g/L (4.6) (range, 18 to 60 g/L [1.8 to 6.0]). Thoracic FAST and Vet BLUE were performed in several dogs ($n = 5$), and no evidence of pleural effusion, pericardial effusion, pneumothorax, or alveolar-interstitial edema was detected. All dogs ($n = 11$) had increased ALT activity above the upper reference limit (range, 385 to 10,000 U/L). However, endpoints were not determined in 4 cases. Reference intervals for ALT varied slightly and were dependent on the specific laboratory used, with a general upper reference limit of 118 to 125 U/L. Eight dogs had either Doppler or oscillometric blood pressure readings. Of these 8, 4 were hypotensive, with a median systolic blood pressure of 70 mm Hg (range, 40 to 140). Hemoconcentration was identified ($n = 5$), with a median PCV or Hct of 54.7% (range, 29.8 to 80). Total plasma protein of the peripheral blood ranged from 35 to 70 g/L (3.5 to 7.0), with a median of 60 g/L (6.0).

Hypoglycemia was identified ($n = 4$) with a median BG concentration of 3.89 mmol/L (70 mg/dL) (range, 2.28 to 8.32 mmol/L [41 to 150 mg/dL]). One dog did not have an initial BG measurement obtained. Hyperlactatemia was noted in all dogs that had plasma lactate evaluated ($n = 6$). Median plasma lactate value was 5.6 mmol/L (range, 4.0 to 15.2). Platelet counts were normal, as defined by individual laboratory reference intervals, in 5 dogs, with a median count of $228.5 \times 10^9/L$ ($228 \times 10^3/\mu L$) (range, 161 to $419 \times 10^9/L$ [161 to $419 \times 10^3/\mu L$]). One dog did not have a platelet count evaluated. The remaining dogs ($n = 5$) had platelet counts below the lower reference interval, with a median platelet count of $142 \times 10^9/L$ ($142 \times 10^3/\mu L$) (range, 133 to $168 \times 10^9/L$ [133 to $168 \times 10^3/\mu L$]). All dogs ($n = 11$) had coagulation parameters evaluated (PT and aPTT). Median PT was 29 seconds (range, 13 to 47 s). Median aPTT values were unavailable, as 1 dog had a prolongation greater than the upper reference limit (range, 86 to > 200 s). Five dogs were deemed coagulopathic.

3.3 | Therapy

Epinephrine^{†††} was administered to 41.7% of dogs ($n = 5/11$), 54.5% of dogs ($n = 6/11$) received an H1R antagonist (diphenhydramine^{##}), 72.7% ($n = 8/11$) received an H2R antagonist (famotidine^{||}), and 54.5% ($n = 6/11$) received a glucocorticoid (dexamethasone sodium phosphate,^{***} prednisone^{†††}). Fresh frozen plasma^{†††,§§§} was administered in 72.7% ($n = 8/11$), and 18% ($n = 2/11$) received allogeneic or autologous red blood cells. One dog received a packed red blood cell transfusion (DEA 1.1 negative packed cells^{††††}), and the second was autotransfused. Other therapies included antimicrobials, proton pump inhibitors, prokinetic agents, antiemetic medications, colloidal support, hypertonic saline, dextrose supplementation, antiarrhythmic

medications, nutraceuticals, phytonadione, vasopressor therapy, and analgesic medications.

3.4 | Outcome

The majority of the dogs (91%) in this retrospective case series ($n = 10/11$) survived and were discharged from the hospital without surgical intervention after a diagnosis of AX and associated hemoperitoneum. The average length of hospitalization was 2.5 days (range, 1 to 5 d). The only non-survivor was euthanized after a 2-day hospitalization, at which time progression to hepatic failure was diagnosed, defined by acute onset of hepatopathy characterized by hyperbilirubinemia and the development of coagulopathy.¹³

4 | DISCUSSION

Definitive criteria for the diagnosis of AX in veterinary medicine remain elusive.¹⁴ The diagnosis of AX has typically been based on history, presenting signs, and physical examination findings, including dermal signs such as urticaria and angioedema, though up to 20% of human patients lack dermal signs,^{15,16} and cutaneous manifestations are not as prevalent in veterinary patients.¹ Ultimately, the response to therapy may be conclusive in the diagnosis of AX.

The systemic effects of AX are initially propagated by the chemical mediators secreted from mast cells and basophils, including histamine, heparin, proteases, and proteoglycans. Subsequent activation of the arachidonic acid cascade via activation of phospholipase A2 promotes synthesis and release of multiple chemokines and cytokines as the AX reaction progresses. The inflammatory and vasoactive chemical mediators that are released cause increased vascular permeability and vasodilation, leading to mixed hypovolemic and distributive shock. The coagulation cascade, complement system, and kallikrein-kinin system are also activated.¹ The propagation and persistence of an inflammatory state has been referred to as the biphasic episode of AX, of which 4.7% (range, 0.4 to 23.3%) of people experience, and likely contributes to the complex acquired coagulopathy in these dogs.¹⁷

Traditional AX therapy includes epinephrine as a first-line agent and aggressive fluid resuscitation. Antihistamines (H1R and H2R antagonists) and glucocorticoids are considered second-line agents. In people, glucocorticoids are purportedly indicated to prevent the biphasic episode of AX by blocking the arachidonic acid cascade. Other described therapies in veterinary medicine include oxygen supplementation, positive inotropic support, and bronchodilators.¹⁴ Vasopressors may be considered for vasodilation refractory to fluid resuscitation. Epinephrine administered as repeat boluses or as a constant rate infusion may be required.¹⁸ Fresh frozen plasma may be indicated to address severe coagulopathy, while red blood cell transfusions are less frequently needed.

In this case series of dogs with AX, the unique canine complication of AX-associated medically treated hemoperitoneum is reported and

described.¹⁹ A case report was recently published that described a dog that developed hemoperitoneum and anaphylactic shock associated with *Hymenoptera* envenomation.³ Notably, no dogs in our series had cutaneous signs of AX or a directly witnessed insect bite or sting. The historical reports from a few dogs raise suspicion for an insect bite or sting, as these patients ($n = 5$) were on a walk or outside and exhibited signs of acute pain in a paw or limb ($n = 2$). A single study in people describes AX-associated hemoperitoneum in a woman undergoing a cesarean section.² The hemoperitoneum was not caused by surgical or procedural hemorrhage but, instead, the presence of general oozing upon re-exploration of the abdomen was identified and a diagnosis of disseminated intravascular coagulation secondary to AX was made. The possible mechanisms of AX-associated hemoperitoneum in dogs involve mediators directly released by mast cells, histamine and heparin, and those factors that are a result of more indirect pathways, BK and PAF.

Previous investigators have documented markedly increased ALT activity and sonographic gallbladder wall striation as hallmarks of AX, though not pathognomonic.¹ Although these 11 cases may not definitively demonstrate the presence or development of a hemoperitoneum to be a hallmark of AX, they do represent a unique pathophysiological consequence. Many emergency clinicians are now performing AFAST examinations as a screening test, and the gallbladder "halo effect" is a commonly sought after finding to help strengthen the argument for a diagnosis of AX. However, in weak or collapsed dogs, acute pericardial effusion/tamponade, right-sided heart failure, and dilated cardiomyopathy may present with acute collapse or weakness with ascites and the gallbladder halo effect as well.^{6,8,20,21} Thus, a standardized approach using point-of-care ultrasound helps to better survey the patient at triage.²²

The additional dogs not included in this specific series ($n = 13$) also had evidence of abdominal effusion, though either abdominocentesis or characterization of the effusion was not performed in these animals, with 1 animal lacking a gallbladder (due to a previous cholecystectomy). Thus, an increased number of dogs may have been diagnosed with hemoperitoneum in the presence of an anaphylactic reaction, had their effusions been sampled and cytologically evaluated. The dog that was excluded due to excessively prolonged PT fulfilled all other criteria of AX; however, hemoperitoneum due to anticoagulant rodenticide toxicosis cannot be definitively ruled out.

The sonographic presence of intra-abdominal fluid in these 11 cases ultimately did not result in a change in therapy specifically for AX. However, if profound and clinically significant anemia is noted along the course of evaluation of an identified AX case, hemoperitoneum should be considered as a possible source. Definitive therapy for AX remains under scrutiny in both human and veterinary medicine. Although glucocorticoids are not recommended as first-line therapy for AX, they are considered a second-line therapy to blunt the biphasic episode of AX that may play a role in the complex acquired coagulopathy seen in these dogs. Moreover, glucocorticoids mitigate mast cell degranulation, thus inhibiting histamine and heparin release into circulation, and indirectly mitigate BK by blocking phospholipase A2 and the arachidonic acid inflammatory pathway.

Limitations of our study include its retrospective case series design and the use of traditional coagulation testing without specific factor analysis or advanced coagulation testing. Standardizations were not made for patient therapy, nor was a control group present. Our case series identified a range of therapeutic regimens based on clinician preference. Definitive recommendations for therapeutic interventions to mitigate the persistent coagulopathy found in several of our dogs cannot be made based on this case series but should be considered a goal for future investigation.

Other possible causes for abdominal effusion in dogs presenting with acute collapse or weakness should be considered during initial triage. Differentials may include hemoperitoneum from a ruptured mass, septic peritonitis, bile peritonitis, uroperitoneum, hepatic failure, pericardial effusion, or acute right-sided congestive heart failure.²³ Ultrasound alone cannot characterize the type of fluid based solely on echogenicity; effusion sample acquisition and fluid analysis are required. The clinical constellation of signs, history, signalment, physical examination, laboratory and imaging findings must all be considered to increase the probability of an accurate diagnosis of AX. As recently described, septic peritonitis and AX patients may present with similar clinical signs, laboratory values, and imaging findings.²⁴ In canine hemoperitoneum, however, challenges arise in differentiating whether the cause of hemorrhage is from a bleeding mass or related to a coagulopathy, with the former scenario requiring surgical intervention and the latter being potentially fatal if surgical intervention is attempted. In this case series, the possibility of hemoperitoneum secondary to coagulopathy caused by anticoagulant rodenticide intoxication is considered low. Anticoagulant rodenticide intoxication is most commonly associated with a 3-fold increase in PT, which was not seen in any of the patients presented here.²⁵ Furthermore, only 27% ($n = 3$) patients received vitamin K1 therapy, with a 91% survival rate overall. Performance of a complete abdominal ultrasound in most patients, and Global FAST in the remainder which, as described, evaluates the liver and spleen for masses, rules out the possibility of a bleeding mass as a source of hemorrhage and surveys the thorax for pleural effusion, pericardial effusion, and pulmonary hemorrhage.¹² We have demonstrated a case series of dogs with hemoperitoneum that were medically treated and survived without surgery. In the event an AX-induced hemoperitoneum is present and misdiagnosed as a surgical problem, at least anecdotally, the decision to perform exploratory surgery can lead to increased morbidity and mortality. The prognostic indications of abdominal effusion accumulation in a dog diagnosed with AX cannot be inferred from this case series. As the spectrum of clinical severity in AX is broad, prognosis must continue to be based on the clinical progression and response to therapy in individual circumstances.

In conclusion, AX is a challenging and multifactorial disease entity. Improved recognition and survival times will be able to be achieved with better definitions of the clinical signs seen and pathophysiological effects on each patient. Interestingly, of our 11 dogs, none presented with dermal signs, a typical hallmark for an allergic reaction, though not consistently observed in AX reactions.¹ The identification of hemorrhagic abdominal effusion in a dog presenting in acute hypovolemic



and distributive shock with increased ALT activity and a gallbladder halo effect should be considered as supporting evidence for AX, once other described causes of hemorrhagic abdominal effusion have been ruled out. These dogs have the potential to be successfully treated medically without surgical exploration, which may be unnecessary and unable to identify a source of bleeding. Further studies are warranted to better determine the exact mechanism for the pathophysiology of this phenomenon of hemorrhage and coagulopathy, as well as to more clearly develop criteria for the diagnosis of AX in veterinary patients. Until other targeted medications become available, traditional first-line therapies of epinephrine and fluid resuscitation should be administered. Second-line therapies of H1R and H2R antagonists and glucocorticoids can be considered, though they will not mitigate the acute reaction. Other therapies, such as blood product administration, should be based on individual patient factors, especially in cases in which AX-related hemoperitoneum is suspected or confirmed.

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CONFLICT OF INTEREST

Dr. Lisciandro is a co-owner of FASTVet.com, a private corporation that provides veterinary ultrasonography training to practicing veterinarians. He also teaches ultrasonography courses for Sound, SonoSite, scil animal care, El Medical, Oncura Partners, and Universal Imaging; and has received ultrasound equipment on loan from SonoSite, Sound, El Medical, Oncura Partners, and scil animal care. He has licensed educational materials to El Medical and has been a paid consultant for Oncura Partners and Vet Imaging; his wife Stephanie is Director of Internal Medicine at Oncura Partners. The other authors declare no conflict of interest.

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ENDNOTES

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† Google Sheets, <https://docs.google.com/spreadsheets>, Google, Mountain View, CA.

‡ Microsoft Office Excel 2010, Microsoft Corporation, Redmond, WA.

§ Doppler Flow Detector, Parks Medical Electronics Inc, Aloha, OR.

¶ Mini Doppler Detector, Jorgensen Laboratories Inc, Loveland, CO.

Alpha TRAK 2, Zoetic Inc, Kalamazoo, MI.

|| Nova Critical Care Xpress, Nova Biomedical Corporation, Waltham, MA.

** Lactate Plus, Nova Biomedical Corporation, Waltham, MA.

†† Nova Critical Care Xpress, Nova Biomedical Corporation, Waltham, MA.

‡‡ Coag Dx Citrate Activated PT, Idexx, Westbrook, ME.

§§ Coag Dx Citrate Activated APTT, Idexx, Westbrook, ME.

¶¶ Epinephrine 1:1000, Vetco Inc, St. Joseph, MO.

Diphehydramine hydrochloride, Mylan Institution LLC, Rockford, IL.

||| Famotidine, West-Ward Pharmaceutical, Eatontown, NJ.

*** Dexamethasone sodium phosphate, Bimeda-MTC Animal Health Inc, Cambridge, ON, Canada.

††† Prednisone, West-Ward Pharmaceuticals, Eatontown, NJ.

§§§ Fresh frozen plasma, BluePearl Blood Bank, Tampa, FL.

§§§ Fresh frozen plasma, Animal Blood Resources, Stockbridge, MI.

¶¶¶ Packed red blood cells, BluePearl Blood Bank, Tampa, FL.

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