

Case Report

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Spontaneous hemoperitoneum and anaphylactic shock associated with Hymenoptera envenomation in a dog

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Abstract

Objective – To describe the clinical presentation of a dog with spontaneous hemoperitoneum associated with anaphylactic shock from Hymenoptera envenomation.

Case Summary – An 8-year-old female neutered Beagle presented as an emergency for acute onset of collapse, hematemesis, and hematochezia. The dog was tachycardic, tachypneic, and hypotensive. Initial assessment revealed a hemoperitoneum with documentation of a nonclotting sanguineous peritoneal effusion with PCV 81% and total protein concentration 65 g/L (6.5 g/dL). No obvious masses were appreciated ultrasonographically during original presentation and cytology of the peritoneal effusion was unremarkable. Peripheral PCV was 67% and total plasma protein concentration was 72 g/L (7.2 g/dL). A bee stinger was located in the right upper lip after presentation and was postulated to be the inciting trigger for the abdominal hemorrhage and shock. The patient was medically stabilized and ultimately made an uneventful recovery. Abdominal ultrasound performed 3 days after discharge revealed a single 0.85 cm hyperechoic liver nodule. Follow-up evaluation at 6 and 18 months after presentation documented a normal physical examination, unremarkable thoracic radiographs, and no change in the original hyperechoic liver nodule.

New or Unique Information Provided – This report details an unusual clinical presentation of anaphylactic shock due to Hymenoptera envenomation in a dog. To the authors' knowledge, spontaneous hemoperitoneum associated with anaphylaxis and bee envenomation has not been documented in the veterinary literature.

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Keywords: anaphylaxis, bee sting, canine, coagulopathy, envenomation, hemoabdomen

Abbreviations

aPTT	activated partial thromboplastin time
FAST	focused assessment with sonography for trauma
CRT	capillary refill time
PT	prothrombin time
RI	reference interval
TPP	total plasma protein concentration

Introduction

Known for immediate onset and rapid progression, the hypersensitivity reaction triggered by insect venom results in considerable morbidity and mortality in people.¹ The prevalence of such hypersensitivity in veterinary medicine is unknown. Stinging insects capable of eliciting anaphylactic reactions are of the order Hymenoptera, which includes the families Apidae (honeybees, bumblebees), Formicidae (ants), and Vespidae (wasps or yellow jackets).² The honeybee is unique because when it stings, the double barbed stinger cannot be retracted by the bee and is subsequently left behind.

Bee venom is one of the most well-known triggers of anaphylaxis, capable of inciting a multifaceted response both immunologic and nonimmunologic in nature. The immunologic mechanisms involve the traditional IgE-dependent pathway and an alternative pathway dominated by IgG and platelet activating factor. One bee sting can deliver approximately 50–140 µg of venom protein.² Nonsensitized individuals can die

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after experiencing massive bee envenomation with a venom protein dose of 2,800 µg per kilogram of body weight.³ However, in the majority of cases, immediate anaphylaxis rather than the venom itself is responsible for most of the reported pathologic sequelae.²

Honeybee venom also contains a wide variety of complex enzymes and peptides capable of triggering severe reactions. Melittin, phospholipase A₂, hyaluronidase, histamine, apamin, and mast cell degranulating peptide are some of the most well-known constituents.^{4,5} Instances of Hymenoptera envenomation resulting in acute kidney injury, coagulopathy, acute lung injury, hemolytic anemia, hepatic and gastrointestinal dysfunction, and cardiovascular collapse have all been reported in veterinary patients.^{6–8} Honeybee venom is reported to be a predominant trigger of anaphylaxis-related hemorrhage in people.⁹

This report documents the successful medical management of a dog with spontaneous hemoperitoneum and bee envenomation. To the authors' knowledge, this is the first account of a spontaneous hemoperitoneum associated with bee venom and anaphylaxis in the veterinary or human literature.

Case Summary

An 8-year-old female neutered Beagle weighing 16.6 kg presented to the emergency service for acute collapse, hematemesis, hematochezia, and tachypnea. The patient was described to be previously healthy. An allergic reaction to a bee sting with facial swelling was reported several years earlier. At the time of presentation, the owners were unaware of any trauma, toxin, or medication exposure. The dog was outside in an enclosed yard prior to the onset of clinical signs. The owners report that after spending a brief period of time outdoors, the dog was observed entering the home very weak and ultimately collapsed with labored, rapid breathing. Several episodes of hematemesis and hematochezia were witnessed prior to admission.

On presentation, the dog was obtunded and in sternal recumbency. Heart rate of 190/min, weak femoral pulses, respiratory rate of 40/min with normal respiratory effort, and normal rectal temperature (38°C [100.1°F]) were recorded. Mucous membranes were muddy with prolonged capillary refill time (CRT) of 3 seconds. Hypotension was noted with a Doppler systolic blood pressure of 70 mm Hg and ECG revealed sinus tachycardia. No integument abnormalities were found during triage. Generalized intermittent abdominal splinting was appreciated with the passage of large volumes of hemorrhagic diarrhea. The severe hematochezia continued for several hours beyond presentation.

Immediate stabilization was addressed with flow-by oxygen, peripheral IV catheter placement, the administration of a total of 80 mL/kg of Plasmalyte-A^a over 60 minutes, 7.2 % hypertonic saline solution^b at 5 mL/kg over 10 minutes, and a 5 mL/kg bolus of hydroxyethyl starch^c (670/0.75). A single dose of IV hydromorphone^d (0.1 mg/kg) was provided out of concern for abdominal discomfort, which appeared to dissipate with resolution of the diarrhea.

While resuscitation efforts were underway, the following diagnostic tests were performed: venous blood gas, point-of-care thoracic, and abdominal ultrasound to evaluate for cavitory effusions, CBC, serum biochemistry profile, prothrombin time (PT), and activated partial thromboplastin time (aPTT). Thoracic and abdominal radiographs, along with an abdominal ultrasound performed by a board-certified radiologist, were strongly advised during initial consultation but were ultimately declined due to financial constraints.

Initial venous blood gas showed a metabolic acidosis. The PCV was 67% (reference interval [RI], 37–55%) and total plasma protein concentration (TPP) of 72 g/L (7.2 g/dL; RI, 52–82 g/L [5.2–8.2 g/dL]). Serum biochemistry panel revealed hyperlactatemia, mild hyperglycemia, marked hyperalbuminemia, hyperglobulinemia, hyperbilirubinemia without evidence of gross hemolysis, and increases in alanine aminotransferase, alkaline phosphatase, and gamma-glutamyl transferase activities. With the exception of a relative erythrocytosis, CBC parameters were within RIs including automated and manual platelet counts. Saline dispersion test was negative for agglutination. Coagulation testing revealed a normal PT of 15 seconds (RI, 11–17 s) and a prolonged aPTT of 142 seconds (RI, 72–102 s). Tables 1 and 2 include details of initial venous blood test results. Urinalysis was acquired via free catch after fluid resuscitation and results were unremarkable with no apparent casts, pigmenturia, or inflammation.

The following findings were noted on abdominal focused assessment with sonography for trauma (FAST¹⁰) performed during triage and stabilization: moderate hypoechoic peritoneal effusion (grade 2/4), no obvious gall bladder abnormalities, and no overt intraabdominal masses. In addition, no pleural or pericardial effusions were appreciated on point-of-care thoracic ultrasound. During initial triage and stabilization, an abdominocentesis was performed and 2 mL of nonclotting sanguineous effusion was aspirated with a PCV of 81% and TPP concentration of 65 g/L (6.5 g/dL). Cytology of the peritoneal effusion had the appearance of peripheral blood devoid of platelets, with no apparent microorganisms, neoplastic cells, or other etiologic agents.

Systemic hypotension, tachypnea, and sinus tachycardia resolved within approximately 90 minutes from

Table 1: Serial complete blood count and coagulation profile results from a dog with Hymenoptera envenomation, anaphylaxis, and spontaneous hemoperitoneum

	Reference Interval	Presentation (time 0)	Hour 4	Hour 10	Hour 24	Hour 36	Hour 120
White blood cell count	$6-17 \times 10^9/L$ ($6-17 \times 10^3/\mu L$)	8.3 (8.3)	N/A	N/A	N/A	20.6 (20.6)	16.6 (16.6)
Segmented neutrophil count	$2.94-12.7 \times 10^9/L$ ($2.94-12.7 \times 10^3/L$)	6700	N/A	N/A	N/A	17304	14110
Band neutrophil count	$0-1.70 \times 10^9/L$ ($0.0-170 \times 10^3/L$)	N/A	N/A	N/A	N/A	1442	N/A
Platelet count	$200-500 \times 10^9/L$ ($200-500 \times 10^3/\mu L$)	275 (275)	N/A	N/A	N/A	251 (251)	273 (273)
Prothrombin time	11–17 s	15	N/A	14	N/A	N/A	N/A
Activated partial thromboplastin time	72–102 s	142	N/A	99	N/A	N/A	N/A
Packed cell volume	37–55%	67	32	30	25	31	55
Reticulocyte count	10–110 K/ μL	N/A	N/A	N/A	N/A	30	169
Total plasma protein	48–76 g/L (4.8–7.6 g/dL)	72 (7.2)	40 (4.0)	42 (4.2)	52 (5.2)	60 (6)	62 (6.2)

Table 2: Serial venous blood and serum biochemistry results from a dog with Hymenoptera envenomation, anaphylaxis, and spontaneous hemoperitoneum

	Reference Interval	Presentation Time 0	Hour 4	Hour 10	Hour 24	Hour 36	Hour 120
pH	7.340–7.420	7.273	N/A	7.366	N/A	N/A	N/A
Lactate	0.3–3.4 mmol/L	7.7 (69.3)	3.0 (27)	3.0 (27)	N/A	N/A	N/A
HCO ₃ [−]	16.0–24.0 mmol/L (16.0–24.0 mEq/L)	12.8 (12.8)	N/A	21.1 (21.1)	N/A	N/A	N/A
Base excess	−2 to +2 mmol/L	−11.5	N/A	−3.1	N/A	N/A	N/A
PvCO ₂	3.25–5.23 kPa (24.4–39.3 mm Hg)	3.65 (27.4)	N/A	4.87 (36.6)	N/A	N/A	N/A
Blood urea nitrogen	9.0–29.0 mmol/L (25–81.2 mg/dL)	10 (28)	N/A	8 (22.4)	N/A	9.64 (27)	10 (28)
Creatinine	35.7–123.7 μ mol/L (0.4–1.4 mg/dL)	79.56 (0.9)	N/A	61.88 (0.7)	N/A	79.56 (0.9)	123.76 (1.4)
Albumin	25–40 g/L (2.5–4.0 g/dL)	50 (5.0)	N/A		N/A	24 (2.4)	31 (3.1)
Globulin	20–36 g/L (2.0–3.6 g/dL)	51 (5.1)	N/A	N/A	N/A	24 (2.4)	31 (3.1)
Alkaline phosphatase	18–121 U/L	376	N/A	N/A	N/A	392	146
Alanine aminotransferase	5–160 U/L	198	N/A	N/A	N/A	217	143
Gamma-glutamyl transferase	0–14 U/L	130	N/A	N/A	N/A	N/A	N/A
Total bilirubin	0–8.55 μ mol/L (0–0.3 mg/dL)	76.95 (4.5)	N/A	N/A	N/A	5.13 (0.3)	1.71 (0.1)

time of presentation with the following parameters recorded: systolic Doppler blood pressure of 132 mm Hg, respiratory rate of 24/min, heart rate of 108/min, improved mentation, and pink mucous membranes with a CRT < 2 seconds. Additional therapies administered after resuscitation included famotidine^e (1 mg/kg IV q 12 h) and ampicillin sulbactam^f (30 mg/kg IV q 8 h). In addition, plasmalyte-A was continued (4.2 mL/kg/hr)

along with hydroxyethyl starch (1.2 mL/kg/hr). The volume of ongoing diarrhea losses were estimated and replaced with an isotonic crystalloid fluid every 1–2 hours.

Four hours after presentation, a bee stinger was identified and removed from the right maxillary lip with mild angioedema and erythema noted at the sting site. After detection of the bee stinger, diphenhydramine^g

(2.4 mg/kg IM q 8 h) was administered. A peripheral PCV of 32% and TPP 40 g/L (4.0 g/dL) was documented and serial lactate measurements had normalized. The patient appeared to remain hemodynamically stable with normal sinus rhythm recorded on continuous ECG, normal respiratory rate and effort, pink mucous membranes with CRT < 2 seconds, and Doppler blood pressure of 140 mm Hg.

Serial abdominal FAST and abdominocentesis 10 hours after presentation revealed a comparable volume of hypoechoic peritoneal effusion (grade 2/4)¹⁰ with an abdominal PCV of 48% and total protein concentration of 45 g/L (4.5 g/dL). The patient's perfusion parameters were normal and the hematochezia subsided considerably with no emesis noted. At this time, with the exception of a low BUN, serial laboratory results were unremarkable including venous blood gas, PT, aPTT, and platelet count (Table 2). Peripheral blood PCV/TPP had declined to 30%/42 g/L (4.2 g/dL).

The patient remained clinically stable and was ambulatory and eating 24 hours after presentation. Recorded vital parameters included a temperature of 38°C (100.5°F), heart rate 130/min, respiration rate 24/min, Doppler blood pressure of 140 mm Hg, and frank blood still present on rectal examination. Ongoing anemia and hypoproteinemia persisted (PCV 25%, TP 52 g/L [5.2 g/dL]) with spherocytes noted on blood smear. Saline dispersion test remained negative.

The patient was discharged 48 hours after presentation with normal physical examination parameters and resolution of gastrointestinal signs. Serial abdominal FAST prior to discharge showed resolving hypoechoic peritoneal effusion (grade 1/4).¹⁰ Instructions were provided for continued administration of diphenhydramine^g and famotidine^e over the following 3 days and discontinue amoxicillin/clavulanic acid^h within 2 days. Serum biochemistry panel and CBC were submitted 48 hours after presentation and showed normalization of total bilirubin concentration and persistent increases in alanine aminotransferase and alkaline phosphatase activities. Hypoalbuminemia, mild hypokalemia, and mild hyponatremia were present. A neutrophilia with slightly toxic changes and a left shift was apparent along with a nonregenerative anemia with a PCV of 27%.

Seventy-two hours after release, the patient returned for a full abdominal ultrasound with a board-certified radiologist, arranged out of professional interest. The patient had been doing well at home with a normal appetite and formed stools. Abdominal ultrasound revealed a single 0.85 cm hyperechoic liver nodule, scant hypoechoic peritoneal effusion, and a mildly hypoechoic pancreas. No other nodules or masses were noted. A CBC and serum biochemistry panel showed reticulocytosis, resolution of anemia, normal platelet count, small-to-

moderate spherocytosis, and markedly improved liver enzyme activities. Serum bilirubin and albumin were normal.

Thoracic radiographs, physical examination, and abdominal ultrasound were also evaluated 6 and 18 months after discharge. Thoracic radiographs and physical examination were within normal limits. Abdominal ultrasound (performed by the same board-certified radiologist) was unremarkable with exception of the previously noted liver nodule, which showed no change in size or echogenicity. The patient was reported to be doing well at home since discharge.

Discussion

Hemoperitoneum is frequently diagnosed in dogs and presents in either spontaneous or traumatic form.¹¹ Spontaneous peritoneal hemorrhage most commonly originates from ruptured neoplasms. Other causes include ruptured masses such as hematomas or granulomas, vessel rupture associated with gastric dilatation-volvulus, torsion of abdominal organs, coagulopathy, and others.¹¹ Additional causes of hemoperitoneum reported in people, but not in dogs, have included variceal rupture linked to portal hypertension with hepatic cirrhosis,¹² ruptured aneurisms, and rare reports of gastric vessel rupture associated with vomiting.¹³

Hallmark characteristics of hemoperitoneum may include cardiovascular collapse, anemia, hypoproteinemia, prolonged aPTT, hyperfibrinolysis, and thrombocytopenia.^{11,14,15} Unique clinical features of this case atypical for spontaneous hemoperitoneum included initial increase in peripheral and abdominal PCV, normal TPP, profound gastrointestinal signs, and cutaneous angioedema associated with a bee stinger.

Bee venom is known to induce anaphylaxis-related hemorrhage and coagulopathies in people, with reports of pulmonary, intracranial, and uterine bleeding.^{6,9} Disseminated intravascular coagulation and thrombocytopenia with secondary hemorrhage have also been reported with Hymenoptera envenomation in a dog.¹⁶ Given the documentation of a bee stinger in the dog in the current report, lack of known trauma and absence of a clear causative intra-abdominal lesion on ultrasound, the hemoperitoneum in this case is suspected to be associated with alterations in hemostasis associated with anaphylaxis and bee envenomation.

The authors hypothesize that derangements in both coagulation and fibrinogenolysis had significant influence in this case due to bee venom components and the anaphylactic response. Such derangements are rarely reported with bee envenomation. The chief constituents of bee venom that can result in coagulopathy and fibrinogenolysis include melittin, phospholipase A₂, and

hyaluronidase.⁶ The peptide melittin is the principal component of bee venom comprising 50% of the dry weight^{4,5} and is responsible for much of the local pain and inflammation.⁴ Melittin is capable of directly activating the bradykinin pathway, which is known to trigger tissue plasminogen activator and plasminogen, ultimately resulting in the formation of plasmin with subsequent development of fibrinogenolysis.⁶ Phospholipase A₂ (comprising 12% of the dry weight) is the primary allergen in bee venom.^{2,5,17} It possesses anticoagulant properties capable of triggering the contact activation system via erythrocyte fragmentation. This results in activation of Factor XII with subsequent release of bradykinin. Hyaluronidase facilitates rapid dispersement of venom components throughout the interstitium,¹⁷ and is reported to exert anticoagulant activity though the mechanism is not well characterized.⁹

Mediators released from mast cells and basophils during anaphylaxis, such as tryptase, histamine, heparin, and platelet activating factor, are capable of influencing fibrinogenolysis, vascular permeability, and coagulation.^{9,18} Anaphylaxis-associated hyperfibrinogenolysis has been documented in the human literature, although infrequently.^{6,18} Tryptase release from mast cells during anaphylaxis induces fibrinogenolysis, which has been documented in people via thromboelastography.^{19,20} Tryptase triggers the cleavage of fibrinogen and release of tissue plasminogen activator, which leads to conversion of plasminogen to plasmin and breakdown of fibrin clots.¹⁹ Concentrations of tryptase and platelet activating factor are shown to increase significantly during anaphylactic reactions.²¹ In fact, blood concentrations of platelet activating factor are shown to have excellent correlation with severity of anaphylactic signs and may directly impact outcome.²¹

Vasculitis secondary to bee envenomation and anaphylaxis has been reported to cause central nervous system hemorrhage in people and could have also played a contributing role in the current patient's hemoperitoneum and gastrointestinal signs.²² Melittin, platelet activating factor, and the bradykinin pathway significantly contribute to vasculitis and interstitial edema.^{6,23} In addition, histamine can lead to the development of vasculitis along with other mediators of anaphylaxis such as heparin and tryptase.²³ The sudden onset of vascular permeability could have intensified this patient's hypovolemia, anemia, and hypoproteinemia. In addition, histamine can promote the production of nitric oxide, which would further contribute to systemic vasodilation and hypotension noted in this patient.²⁴

The aPTT was increased initially in this case and normalized spontaneously. Possible causes for the dog's increased aPTT include anaphylaxis-associated heparin release, consumptive coagulopathy that may

have been exacerbated by erythrocyte pore formation, and dilutional coagulopathy. Heparinization secondary to mast cell degranulation and anaphylaxis may have contributed to increased aPTT in this dog, although this mechanism of hypocoagulability is rare *in vivo*.^{19,20} Heparin acts as an anticoagulant by binding to antithrombin and promoting the inactivation of thrombin.¹⁹ In humans, reports of heparinization secondary to anaphylaxis induced by Hymenoptera envenomation have been documented with increased anti-Xa concentration, increased aPTT, and decreased fibrinogen concentration in the face of normal PT and platelet count.¹⁹

Consumptive coagulopathy is a noted complication of both Hymenoptera envenomation and anaphylaxis in the dog.¹⁶ In a clinical practice setting, a combination of PT, aPTT, platelet count, plasma fibrinogen concentration, and plasma d-dimer concentration are used to make a presumptive diagnosis of disseminated intravascular coagulation.²⁵ The absence of thrombocytopenia and lack of persistent prolongation of aPTT or PT failed to support consumptive coagulopathy in this dog; however, no single test is capable of confirming or rejecting this diagnosis.²⁵ In the current case, clotting times normalized and clinical hemorrhage resolved without coagulation factor replacement.

Hemolysis secondary to the formation of erythrocyte pores is characterized by spherocytosis on blood smear and could account for the spherocytes noted in this case. Phospholipids are fundamental to the integrity of cell membranes and mitochondria. Melittin, exogenous phospholipase A₂, and hyaluronidase have all been shown to promote erythrolysis.^{5,9} Melittin is also capable of stimulating endogenous phospholipase A₂, which increases cell membrane permeability.^{5,9} Production of cell fragments from pore formation can lead to activation of Factor XII and initiation of the contact pathway. This can result in a self-limiting consumption of coagulation factors and temporary coagulopathy, transient prolongation of aPTT, and the potential for inappropriate hemorrhage.⁹ This mechanism of erythrocyte destruction should not be confused with reports in dogs of immune-mediated hemolytic anemia associated with prior bee envenomation.⁷ With normalization of serum total bilirubin concentration, no evidence of pigmenturia, and negative saline dispersion test, an antigen-antibody immune-mediated hemolytic process was considered unlikely in this case.

Dilutional coagulopathy occurs most frequently in cases requiring significant fluid resuscitation, often incorporating crystalloids, synthetic colloids, or packed red blood cells.²⁶ The most common hemostatic changes include thrombocytopenia, prolongation of both PT and aPTT, and hypofibrinogenemia.²⁷ In this dog, initial blood samples were obtained during the initiation of

a crystalloid fluid bolus and prior to the administration of synthetic colloid, rendering dilution an unlikely cause of aPTT prolongation here.

Complete interrogation of this dog's primary and secondary hemostatic systems, endogenous anticoagulant profile, and fibrinolytic systems may have helped to explain its spontaneous hemoperitoneum. However, tests in this case were limited to on-site evaluation of PT, aPTT, and platelet count. Measurement of plasma fibrinogen, antithrombin, d-dimer, and activated protein C concentrations; individual coagulation factor analysis; and viscoelastic testing would likely have better explained this dog's bleeding tendency.

Initial clinical signs in this dog appeared consistent with hypovolemic shock secondary to peritoneal and gastrointestinal hemorrhage. It was later recognized that profound vasodilation and vasculitis secondary to anaphylaxis were significant contributors to cardiovascular collapse. Earlier identification of distributive shock from anaphylaxis and vasopressor use may have resulted in less aggressive fluid requirements and more rapid stabilization in this case.²⁸ The benefits of epinephrine during anaphylaxis include vasoconstriction, positive inotropy, and bronchodilation.²⁸ Epinephrine is also known to inhibit the release of anaphylactic mediators such as histamine from mast cells and basophils.²⁸

Portal hypertension secondary to anaphylaxis likely played a significant role in this patient's shock. Studies have shown that hepatic vascular resistance can transiently increase due to acute hepatic vasoconstriction associated with histamine release during anaphylaxis.²⁹ Portal hypertension can lead to both hepatic and visceral congestion and result in splanchnic pooling of blood within the gastrointestinal tract²⁴; such congestion may have contributed to the hematochezia and emesis seen in this dog. Portal hypertension can also significantly diminish cardiac return and negatively impact cardiac output, which may have exacerbated the hemorrhagic, hypovolemic, and distributive shock in this dog.

The ultrasound finding of a single 0.85 cm hyperechoic liver nodule was documented to be unchanged 18 months post-discharge. Potential differentials for the hyperechoic hepatic nodule include nodular hyperplasia, myelolipoma, benign neoplasia, granuloma, dystrophic mineralization, primary or metastatic neoplasia, or abscessation.³⁰ A malignant lesion such as hemangiosarcoma was unlikely given the patient's survival time, lack of recurrent clinical signs, initial ultrasonographic appearance of the nodule (typically hypoechoic to heterogeneous and cavitary),³⁰ and absence of ultrasonographic change in the nodule. Given these facts, while histopathologic evaluation of the lesion would have been ideal, the lesion is not considered to have played a role in this dog's spontaneous hemorrhage.

Conclusion

The underlying cause of this dog's suspected bleeding disorder was likely multifactorial and attributable to acute hyperfibrinogenolysis, coagulopathy, and vasculitis associated with severe anaphylaxis and bee venom elements. Hemoperitoneum associated with anaphylaxis may be mistakenly attributed to hemorrhage alone, and the severe peripheral vasodilation and vascular permeability secondary to anaphylaxis may go unrecognized. Hymenoptera stings have been documented to cause internal hemorrhage in people and should be recognized as a rare, potential cause of hemoperitoneum in dogs.

Footnotes

- ^a Plasmalyte-A, Abbott Laboratories, North Chicago, IL.
- ^b Hypertonic saline, MWI, Boise ID.
- ^c Hetastarch, Hospira, Inc, Lake Forest, IL.
- ^d Hydromorphone, West-ward.
- ^e Famotidine, Mylan Institutional LCC, Rockford, IL.
- ^f Ampicillin sulbactam, Aurobindo Pharma LLC, Dayton, NJ.
- ^g Diphenhydramine, West-ward, Eatontown, NJ.
- ^h Amoxicillin/Clavulanic acid, Zoetis, Kalamazoo, MI.

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