CASE REPORT

Non-coagulopathic hemothorax in a dog: A case report

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Abstract
An 8-year-old, female-spayed, Blue Heeler presented with suspected non-coagulopathic spontaneous, hemothorax with neoplasia as the most likely differential as coagulation parameters were normal and no obvious history of trauma was reported. Computed tomography scan was crucial for diagnosis of a traumatic partial vascular avulsion injury that was successfully managed supportively.

KEYWORDS
canine, hemothorax, pleural effusion, vascular avulsion

1 | CASE PRESENTATION

1.1 | Day one

An 8-year-old, female-spayed, 15.1 kg Blue Heeler was presented to the Veterinary Teaching Hospital emergency service for a suspected snake envenomation after the patient momentarily ran out of view and vocalized during a hike. The owner had noted a snake in the patient's path prior to this incident making a snake bite a distinct possibility. The patient returned to the owner acutely ataxic and lethargic. On presentation, the patient was ataxic, weak, tachypneic (60 breaths per minute) with pale pink mucous membranes. The heart sounds were muffled bilaterally with decreased lung sounds. An area of non-painful, marked ecchymosis was present on the left ventral aspect of the patient's neck. There was an approximately 4 mm central indentation with suspected devitalized tissue. A line of demarcation was made with a marker around the area of ecchymosis. A thoracic point-of-care ultrasound (POCUS; Chison Medical Technologies Sound Wave 5 Vet, Chison, ultrasound) was performed which revealed bilateral pleural effusion with a hyperechoic structure in the dog's pleural space adjacent to the heart. A complete blood count (Sysmex XN-V-1000, Sysmex America), small animal chemistry profile (Beckman Coulter, AU 480), prothrombin time (PT; Sysmex CA-660, Siemens Healthcare Diagnostics), partial thromboplastin time (PTT; Sysmex CA-660, Siemens Healthcare Diagnostics), and whole blood lactate (Lactate plus, Nova biomedical) were performed. The patient's complete blood count at presentation revealed a low plasma protein of 5.0 g/dl (RI 5.9–8.2 g/dl) with a normal hematocrit (HCT) of 40.4%. The PT and PTT were unremarkable. The lactate was elevated at 4.9 mmol/L (RI <1.6 mmol/L). The chemistry changes are listed in Table 1.

The patient had a hemoglobin saturation level of 93% to 94%, as determined by pulse oximetry (Rad-57 Handheld Pulse Oximeter-9216-U, Masimo) (SpO2). The patient’s oscillometric blood pressure (Cardell Veterinary monitor model 9405, Midmark Corporation) at presentation was 84/56 (69) mmHg. This was repeated 30 min later revealing a blood pressure of 72/38 (54) mmHg. The patient received a 10 ml/kg bolus of Lactated Ringer’s Solution (LRS; Lot JOD169. BBran/Mcgaw) intravenously. The patient continued to receive intravenous LRS at 50 ml/hr. Pain medication was initiated with buprenorphine (Par Pharmaceuticals) (0.01 mg/kg IV q 8 hours). A left-sided thoracentesis was performed and 10 ml of hemorrhagic, non-clotting effusion was removed.
A HCT/total protein (TP) of the sample was obtained. The HCT (HemataSTAT-II, Separation Technology, INC.) of the effusion was 47.3% (RI 37.3%– 61.7%) and TP was 6.2 g/L (RI 5.9– 8.2 g/L). The patient was blood typed and crossmatched in the event a transfusion was necessary. Aminocaproic acid (Hospira) (50 mg/kg IV q8 hours for 24 h) was initiated. The dog was placed in an oxygen cage set at 40% oxygen and monitored with telemetry overnight.

The ecchymosis continued to spread past the line of demarcation from intake. A repeat PT/PTT was unre markable revealing a PT of 7.5 s (RI 7.3– 8.8 s) and PTT of 9.6 s (RI 9.9–12.1 s). A HCT/TP was performed and was consistent with blood loss anemia (23%/4.6 g/L). A plasma transfusion was administered over 4 h. During the plasma transfusion, the patient exhibited sustained runs of ventricular premature complexes and a lidocaine bolus (Vet One) (2 mg/kg IV) was administered, followed by a constant rate infusion (CRI) (50 mcg/kg/min). A dose of maropitant (Zoetis) (1 mg/kg SQ) was administered after starting the lidocaine. The patient developed a mild plasma transfusion reaction that resulted in swelling around the lips and right lower eyelid. The accelerated idioventricular rhythm continued and lidocaine was discontinued 4 h later.

### Day two

Serial HCT/TP measurements were performed throughout the day and the HCT maintained between 17.9 and 25%. A packed red blood cell transfusion was performed once the HCT dropped to 17.9% and the respiratory rate increased to 60 breaths per minute.

The patient was anesthetized and underwent a computed tomography (CT) scan (Toshiba Acquilion 16, Canon Medical Systemas USA, Inc.) of the neck and thorax (Figure 1). Sequential transverse images of the thorax and abdomen (1 mm slice thickness) were taken before and 3 and 10 min after administration of 33 ml of Omnopaque 300 (GE Healthcare). A large quantity of pleural effusion (pre-contrast 44 HU, post-contrast 43 HU) was noted within the left pleural space with a smaller quantity in the right pleural space. There were multiple variably-sized amorphous regions within the pleural space that were hyperattenuating to the surrounding pleural effusion (pre-contrast 63 HU, post-contrast 64 HU) consistent with acute clotted hemorrhage (sentinel clot sign).

There were segments of the left internal thoracic artery and caudal circumflex humeral vein that were void of contrast (appearing discontinuous) and surrounded by conforming hyperattenuating material of similar characteristics to acute clotted hemorrhage (Figure 2). The patient had minimal left-sided pneumothorax.

Based on the CT findings, a disruption of the left internal thoracic artery and/or the left caudal circumflex humeral vein was diagnosed as the likely etiology of the bilateral pleural effusion with concurrent cranial mediastinal hemorrhage/edema. The hyperattenuating, non-enhancing amorphous material likely represented clotted blood surrounded by hemorrhagic effusion.

The patient recovered uneventfully from anesthesia. A second lactate was performed revealing resolution of hyperlactemia 0.98 mmol/L (RI <1.6 mmol/L). Conservative management, including rest and oxygen, was pursued with the understanding that surgery or additional transfusions might become necessary.

### Day three

The patient remained stable in hospital and showed an improved HCT/TP of 38%/7.6 g/dl. The patient did not exhibit increased respiratory effort and average respiratory

| Variable   | Result          | Reference interval       |
|------------|-----------------|--------------------------|
| Glucose    | 6.88 mmol/L     | 4.88–6.72 mmol/L         |
| Creatinine | 129.95 μmol/L   | 61.88–114.92 μmol/L      |
| Phosphorus | 1.71 mmol/L     | 0.61–1.42 mmol/L         |
| Calcium    | 4.3 mmol/L      | 4.7–5.35 mmol/L          |
| Total protein | 41 g/L     | 53–70 g/L                |
| Albumin    | 23 g/L          | 28–37 g/L                |
| Globulin   | 18 g/L          | 21–38 g/L                |
| ALT        | 13 U/L          | 16–75 U/L                |
| Total bilirubin | 1.71 μmol/L    | 3.42–6.84 μmol/L         |
| Total carbon dioxide | 16 mEq/L      | 18–25 mEq/L              |
| Sodium     | 141 mmol/L      | 143–152 mmol/L           |
rates were 23 breaths per minute. The patient was discharged from the hospital on Day 3 with a HCT/TP of 34.7%/7.4 g/dl. Medications dispensed included carprofen (Zoetis) (2.4 mg/kg PO q12 hours) and trazodone (Teva Pharmaceuticals) (3.3 mg/kg PO q8-12 h). Strict exercise restriction was recommended for 14 days.

1.4 | Day five and beyond

The patient was presented for a scheduled recheck on Day 5. The patient’s vital parameters were normal and the HCT/TP continued to improve (47%/8 g/dl). Two months after the incident, the patient was presented to the veterinary teaching hospital community practice for an annual wellness examination and vaccination. The patient was reported to be doing very well at home with no observed abnormalities reported by the client.

2 | DISCUSSION

This report describes a case of a dog with a non-coagulopathic hemothorax due to an unwitnessed traumatic event. Initial concerns for this patient included hypovolemic shock and hemothorax secondary to a venomous snake bite, based on the compatible historical information and examination findings. However, upon examination, the area of ecchymosis was non-painful and deemed unlikely to be the result of a snakebite.

The patient’s pleural effusion was initially suspected based on auscultation of muffled heart sounds, decreased lung sounds and tachypnea. Tachypnea can be recognized after accumulation of 20 ml/kg of pleural fluid. A rapid diagnosis of pleural effusion can be established with thoracic POCUS. The patient had a large hyperechoic structure in the pleural space adjacent to the heart that was presumed to be a mass or hematoma. In retrospect, the hyperechoic structure seen on POCUS was likely to be clotted blood as suggested by the CT scan.

Since the patient was not severely compromised by the pleural effusion, the decision was made to stabilize hypovolemic shock and await assessment of biochemical, blood cell count and coagulation parameters prior to thoracentesis. The diagnosis of a hemothorax is defined as the accumulation of blood in the pleural space with a packed cell volume (PCV) that is at least 25% of peripheral blood. The clinical sequelae of a hemothorax are more likely secondary to hypovolemia from blood loss as opposed to pleural effusion. It has been suggested that in canine patients hemothorax does not require routine drainage of the hemorrhagic fluid. A generally accepted indication for drainage is to improve respiratory effort in patients with compromised breathing that is believed to be caused by the presence of pleural effusion. On CT scan, the patient had a minimal left-sided pneumothorax. A traumatic pneumothorax is considered unlikely given the minimal volume.

The causes for non-coagulopathic hemothorax include trauma and spontaneous etiologies. The most common traumatic injuries reported are road traffic collision, penetrating injury, crush/compression injury, acceleration or deceleration injury and iatrogenic. The evidence of possible trauma for our patient was the history of running out of view and yelping prior to returning to the owner and the area of ecchymosis with focal area of suspected devitalized tissue. A traumatic hemothorax was initially deemed less likely given the patient was only out of view momentarily with no obvious cause for the trauma, nor lacerations. A through and through bullet wound is considered unlikely given the owner did not hear a gunshot and the wounds on the skin were not suggestive of a bullet wound. In hindsight, given no neoplasm was found on CT, blunt force trauma or impalement are deemed most likely causes for this patient’s hemothorax.

Spontaneous, non-coagulopathic causes of hemothorax include neoplasia (osteosarcoma, hemangiosarcoma,
mesothelioma, pulmonary carcinoma, and metastatic ovarian carcinoma), lung lobe torsion, infectious diseases (Dirofilaria immitis, Streptococcus zooepidemicus, Angiostrongylus vasorum and Spirocerca lupi), thymic hemorrhage, Ehlers–Danlos syndrome and pancreatitis.

A study of 16 client-owned dogs with non-coagulopathic, spontaneous hemothorax demonstrated that neoplasia was the most common cause. Given the patient’s age, concurrent hemorrhagic effusion and absence of known trauma, the possibility of neoplasia was discussed with the owner prior to proceeding with further diagnostics. It is possible that with a less committed owner the discussion of possible neoplasia could have led to the undue euthanasia of a patient that would have otherwise recovered with supportive management.

The patient was prescribed carprofen, a non-steroidal anti-inflammatory medication. This medication has increased risk of kidney damage in the face of azotemia. A repeat chemistry panel would have ideally been performed prior to discharge with carprofen to evaluate the patient’s kidney values. The patient had a repeat chemistry panel 13 months after discharge with the primary veterinarian that did not reveal continued azotemia.

Aminocaproic acid was utilized as an antifibrinolytic drug to decrease the breakdown of clots to reduce hemorrhage and subsequent need for transfusion. In a single retrospective study of 122 dogs with hemorrhage of various causes that were treated with aminocaproic acid, the drug was generally well tolerated, but a clear benefit for its use was not established. Neoplasia was the cause of bleeding in the majority of dogs in that study while only 6 dogs had bleeding secondary to trauma. The aminocaproic acid dose ranged from 9 to 111 mg/kg in this retrospective study. It was elected to administer a 50 mg/kg dose given the large reported dose range. Antifibrinolytic drugs have shown promise in reducing bleeding in human patients with trauma, yet it remains unclear if similar treatments may be beneficial in canine trauma patients.

The patient received a plasma transfusion overnight due to increased concern for snake envenomation. The most common venomous snakes seen in Southwest Virginia are the eastern copperheads (Agkistrodon contortrix) and the timber rattlesnakes (Crotalus horridus). In retrospect, the plasma transfusion was likely unnecessary as there was no documented coagulopathy. In trauma cases, a plasma transfusion may be indicated if massive transfusion is needed. In one study of human trauma patients who did not meet the criteria for massive transfusion, transfusions of fresh frozen plasma were not shown to confer a survival benefit, and in fact were associated with more complications. This patient did have a mild transfusion reaction, and although no severe adverse events were noted, the plasma transfusion was likely unnecessary.

The patient was administered intravenous lidocaine followed by a lidocaine CRI after the heart rate increased to 130 beats per minute with runs of ventricular premature complexes. The lidocaine CRI was continued for 4 h overnight without significant improvement. Given the patient was having accelerated idioventricular rhythm and not ventricular tachycardia, the lidocaine was not necessary for arrhythmic control but could have contributed to analgesic support.

The patient was presented outside of normal business hours and was stabilized prior to advanced imaging the next morning. A CT scan with contrast was critical to the diagnosis of the vascular avulsion injury. CT has been shown to be more sensitive than radiography in evaluation of the trauma patient. CT has been shown to detect a larger percentage of cases with pulmonary contusion, pleural effusion or pneumothorax when compared to radiographs. In addition, post-contrast CT images provide exceptional detail of vascular anatomy that rivals the more invasive contrast angiography.

3 | CONCLUSION

This case report highlights the importance of considering multiple differentials for an unclear etiology of hemothorax. Neoplasia was our top differential for a suspected spontaneous, non-coagulopathic hemothorax and could have led to unnecessary euthanasia of the patient. A CT scan was critical to the diagnosis of vascular avulsion injury secondary to trauma and development of a conservative therapeutic plan for the patient.

AUTHOR CONTRIBUTIONS

DL, MF, and BC were part of the patient’s healthcare team. DL, MF, BC, and GD wrote the manuscript. All authors reviewed and approved the final version of the manuscript.

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None.

CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.
CONSENT
Written informed consent was obtained from the patient to publish this report in accordance with the journal’s patient consent policy.

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