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Presumptive spontaneous hemothorax associated to thymic involution in a dog with steroid responsive meningitis-arteritis (SRMA)

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ABSTRACT

Objective: To describe an unusual case of spontaneous hemothorax resulting from thymic involution in a dog with suspected acquired bleeding dyscrasia associated with steroid-responsive meningitis-arteritis (SRMA).
Case description: A 6-month-old spayed female Golden Retriever was referred due to the sudden onset of lethargy, fever (pyrexia), loss of appetite (anorexia), and moderate neck pain. These symptoms emerged six days after an ovariohysterectomy performed by the primary veterinarian. Upon admission, the patient exhibited pale mucous membranes, tachycardia (180 bpm), bilateral muffled heart sounds and tachypnea. Abdominal and thoracic point-of-care ultrasound (POCUS) were performed and revealed bilateral pleural effusion. Due to the patient's unstable condition, emergent thoracocentesis and transfusion of packed red blood cells was required. The initial work-up performed included a complete blood cell count (CBC), biochemistry profile, venous blood gas and coagulation panel (PT, APTT, fibrinogen). Pleural effusion analysis was compatible with hemothorax. Bloodwork was unremarkable including the initial coagulation panel. Further coagulation test was performed including buccal mucosal bleeding time, viscoelastic-based clot detection tests (TEG) and Von Willebrand factor antigen measurement. TEG revealed marked hyperfibrinolysis. *Angiostrongylus* vasorum and 4DX snap test were performed and yielded a negative result. Thoracic CT scan revealed the presence of a soft tissue-attenuating mass in the ventral mediastinum, thymic involution, and enlargement of the sternal and mediastinal lymph nodes. Therapy with tranexamic acid and corticosteroids at anti-inflammatory doses was initiated. Marked clinical improvement was observed within 24 hours, and after three days of hospitalization the patient was discharged. One month later, the dog was referred again for acute pyrexia, hyporexia, and neck pain which progressed to non-ambulatory tetraparesis. Neurological examination was compatible with C6-T2 lesion. MRI and cerebrospinal fluid analysis were performed and revealed a final diagnosis of steroid-responsive meningitis-arteritis (SRMA) with associated intramedullary hemorrhage. Corticosteroids were started again, and the patient showed a dramatic improvement over the next 24 hours. Three weeks after the diagnosis, the dog returned to a clinically normal state. The treatment was gradually tapered over the following months, guided by regular neurological and clinical examinations and CRP measurements, without any relapses.
New or unique information: To the best of the author's knowledge, this is the first documented case of a dog experiencing spontaneous hemothorax as a result of thymic hemorrhage/involution which, in the absence of other identifiable diseases, was attributed to a hyperfibrinolytic state induced by a severe inflammatory disease such as SRMA.

Introduction

Hemothorax is characterized by the presence of hemorrhagic effusion within the mediastinal space or pleural cavity. Hemorrhagic thoracic effusion can be broadly categorized as traumatic or non-traumatic (spontaneous). They are both characterized by effusion with a packed cell volume (PCV) that is at least 25% of that in peripheral blood.[1] In dogs, spontaneous hemothorax has been associated with either local factors (non-coagulopathic hemothorax) or, more commonly, systemic disorders affecting primary and secondary hemostasis. While the prevalence of non-coagulopathic spontaneous hemothorax in dogs remains unknown and is rarely reported in the veterinary

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literature, case reports have documented its association with conditions such as dirofilariasis, spirocercosis, angiostrongylosis, intrathoracic tumors, diaphragmatic hernia, and pulmonary thromboembolism, among others.[1-3] On rare occasions, non-coagulopathic spontaneous hemothorax has been associated with vascular pathologies or spontaneous rupture of intrathoracic masses or tissues, such as the thymus.[4,5] However, thymic hemorrhage is often diagnosed during postmortem examination due to its high fatality rate[4,6] In young dogs, this condition typically occurs in association with thymic involution, which leads to increased fragility of the vascular bed and a lack of support from adjacent fibro-adipose tissue.[6] There is growing evidence that steroid-responsive meningitis-arteritis is an immune-mediated disease that may also involve vascular beds in organs beyond the nervous system,[5] to the best of the author's knowledge, the association between these two emergent disorders has not been reported in dogs.

Case description

A 6-month-old, female spayed, 20 kg Golden Retriever was referred to the Veterinary Teaching Hospital of Universitat Autònoma de Barcelona for evaluation of acute onset of lethargy, pyrexia, anorexia, and light neck pain. Six days before presentation the dog was spayed at the primary vet and prescribed non-steroidal anti-inflammatory, antibiotic, and analgesic drugs for at home administration. No other relevant clinical information was reported by the owners on admission.

Physical examination abnormalities on admission revealed depressed mentation, weakness, tachypnea (80 rpm), sinus tachycardia (180 bpm), muffled heart sounds on auscultation, hyperdynamic pulse pressure, discomfort on abdominal palpation and light neck pain. Systolic arterial blood pressure obtained by doppler (Ultrasonic doppler flow detector, Parks Medical electronics Inc. Aloha, OR, USA) over the radial artery was 130 mmHg. Calculated shock index was 1.38 (RR < 1). A quick lead two electrocardiogram confirmed the sinus tachycardia and did not identify any other arrythmia. Pulse oximetry (SPO2) measurement revealed a severe hypoxemia of 85%. Abdominal and thoracic point-of-care ultrasound (POCUS) evidenced bilateral pleural effusion. Oxygen flow-by supplementation at 5 L/min was initiated and a right cephalic intravenous catheter was placed. A blood sample was taken from the catheter and the following test were performed: complete blood cell count, biochemistry panel, coagulation panel (PT, APTT, and fibrinogen) and venous blood gas (Tables 1,2,3,4). Packed red cell volume (PCV) was 20%, total solids (TS) 4.5 g/dl and blood glucose was 177 mg/dL (9.52 mmol/L; RR: 80-120 mg/dL [4.44-6.66 mmol/L]). CBC results showed a marked normocytic, normochromic nonregenerative anemia, moderate neutrophilic leukocytosis, and platelet count within reference range. The biochemistry and coagulation profile were unremarkable.

Table 1
Results of hematology.

TEST REQUESTED	RESULTS	REFERENCE RANGE	UNITS
RBC (Red blood cell count)	2.55	5,5 - 8,5	x106/µl
Hgb (Hemoglobin)	5.9	Dec-18	g/dL
Hct (Hematocrit)	17	37 - 55	%
MCV (Mean corpuscular volume)	69.4	62 - 77	fl
MCH (Mean corpuscular hemoglobin)	23.1	21,5 – 26,5	pg
Reticulocyte count	11,475	0 – 60.000	
White blood cell count	21,380	6.000 – 17.000	x/µl
Band neutrophils	214	0 - 300	x/µl
Segmented neutrophils	15,180	3.000 – 11.500	x/µl
Lymphocytes	4,062	1.000 – 4.800	x/µl
Monocytes	1,924	150 – 1.350	x/µl
Eosinophiles	0	100 – 1.500	x/µl
Basophils	0	0 - 200	x/µl
Platelet count	375	200 - 500	x103µl

Table 2
Results of biochemistry.

TEST REQUESTED	RESULTS	REFERENCE RANGE	UNITS
ALT (Alanine aminotransferase)	24	8 – 75	U/L
Total bilirubin	0.2	0 – 0,8	mg/dL
Albumin	2.4	2,1 – 3,6	g/dL
Globulins	4.4	2,5 – 4,5	g/dL
Total protein	6.8	5,2 – 8,2	g/dL
BUN (Blood urea nitrogen)	18	Jul-29	mg/dL
Creatinine	0.6	0,3 – 1,2	mg/dL
Glucose	117	74 - 143	mg/dL
Lactate	0.5	0,5 – 2,5	mmol/L
Magnesium	1.9	1,20 – 2,04	mg/dL
Chloride	109	105 - 119	mmol/L
Sodium	147	145 - 157	mmol/L
Potassium	3.9	3,5 – 5,5	mmol/L

Table 3
Results of coagulation.

TEST REQUESTED	RESULTS	REFERENCE RANGE	UNITS
Prothrombin time (PT)	6.5	6 – 8	Sec
Activated partial thromboplastin time (aPTT)	12.7	Sep-16	Sec
Fibrinogen	422	200 - 400	mg/dL

Table 4
Results of venous blood gases.

TEST REQUESTED	RESULTS	REFERENCE RANGE	UNITS
pH	7.428	7,31 – 7,42	
pCO_2	32.2	32 - 49	mmHg
pO_2	99.1	24 - 48	mmHg
SO_2	100	93 - 100	%
Hct	19	40,3 – 60,3	%
tHb	5.7	Dec-18	g/dL
Na^+	143.4	144 - 160	mmol/L
K^+	3.98	3,5 – 5,8	mmol/L
Cl^-	116.8	109 - 122	mmol/L
iCa	1.35	1,25 – 1,5	mmol/L
iMg	0.6	0,43 – 0,6	mmol/L
Glu	118	65 - 112	mg/dL
Lac	0.3	Low – 2	mmol/L
Creat	0.48	0,7 – 1,8	mg/dL
Urea	23.2	Sep-33	mg/dL
TCO_2	22.4	22 - 26	mmol/L
BE-ecf	-3.1	(- 4) – (+ 4)	mmol/L
HCO_3^-	21.4	20 – 29 mmol/L	mmol/L
paO_2/FiO_2 Ratio	473.9	> 400-500	mmHg

Initially a 20 ml/kg fluid bolus of a balance isotonic crystalloid solution therapy was administered (Lactated Ringer's, B.Braun Medical S. A., Barcelona, Spain) and patient response was monitored. Bilateral thoracocentesis was performed under light sedation with butorphanol (0.2 mg/kg/IV). A total of two hundred and eighty milliliters of a hemorrhagic effusion were retrieved and the patient showed a marked hemodynamic improvement. After procedure, oxygen supplementation was continued via unilateral nasal cannula since SPO2 remained at 92% without oxygen therapy. After thoracocentesis, venous blood gas analysis revealed mild respiratory alkalosis with lactate levels within the reference range. Evaluation of thoracic effusion by a board-certified clinical pathologist confirmed the diagnosis of hemothorax based on a packed cell volume (PCV) of 30%, total solids (TS) of 6 mg/dL, and a cell count of 7200 cells/µl, with 65% non-degenerate neutrophils consistent with an exudative effusion. No infectious agents or neoplastic cells were detected. Based on these findings, supplementary coagulation assessments were performed, encompassing a buccal mucosa bleeding time

(BMBT), a viscoelastic-based clotting test (thromboelastography-TEG), and the quantification of Von Willebrand factor antigen (vWfa). Notably, both BMBT (1.5 minutes, with a normal range of < 3-4 minutes in dogs) and vWfa (73.6%, deemed normal at > 70%) fell within the established reference ranges. However, TEG results revealed a significant hyperfibrinolytic pattern (LY30 40.7%, reference range: 0-2%) (Fig. 1). Based on the TEG results, serological testing for Angiostrongylus vasorum and a snap test were performed, along with a 4DX snap test, all of which returned negative results.

The patient was admitted into the intensive care unit and the following treatments were started: intravenous fluid-therapy with Lactated Ringer's solution supplemented with KCl, and tranexamic acid (10mg/kg/IV/tid). Analgesia, in the form of intravenous methadone (0.2 mg/kg), was administered on an as-needed basis, guided by serial assessments utilizing the Glasgow Pain Scale. This was implemented in response to cervical pain and the thoracocentesis procedure. Oxygen therapy was continued via nasal cannula. Hemodynamic and respiratory parameters were continuously monitored over the first 24 hours. Packed cell volume, serum total proteins, and venous blood gas with electrolytes were monitored every 6 hours during hospitalization or as needed based on the patient's condition. Thoracic radiographs were taken after the initial thoracocentesis and revealed a diffuse and generalized alveolo-interstitial pattern compatible with atelectasis and a poorly defined structure of tissue opacity in the cranio-ventral mediastinum, which was compatible with the thymus gland. During the initial 12 hours following admission to the ICU, the patient maintained stability. Nevertheless, a subtle yet steadily deteriorating respiratory pattern, coupled with an elevated intolerance to manipulation, became apparent over the subsequent day. A subsequent thoracic POCUS confirmed the recurrence of pleural effusion. Also, PCV/TS had dropped to 17% and 4 g/dL respectively. Thoracocentesis was repeated. The patient was transfused with 12mL/kg of compatible DEA 1-negative packed red blood cell. After stabilization, a thoracic CT scan was performed, revealing residual pleural effusion, isolated secondary lung atelectasis, a soft tissue-attenuating mass in the ventral mediastinum compatible with hematoma, severe thymic involution, and sternal and mediastinal lymphadenomegaly (Image 1). Prednisone 1mg/kg/SID was added to the initial plan, and the patient was monitored over the next days in the ICU. No further episodes of hypoxemia occurred and the dog steadily improved. TEG was repeated forty-eight hours after admission showing a normal coagulation trace. Therefore, tranexamic acid was discontinued. Three days after admission in the ICU the patient was discharged under her owner's care. Prednisone was continued for a week.

One month later, the dog was referred back for acute pyrexia, hyporexia and neck pain that progressed over three days to non-ambulatory tetraparesis. The patient's general physical examination revealed fever of 40°C, significant cervical pain, and an irregular heart rhythm on cardiac auscultation, which was confirmed on an ECG and was suggestive of isolated ventricular premature complexes. Abdominal

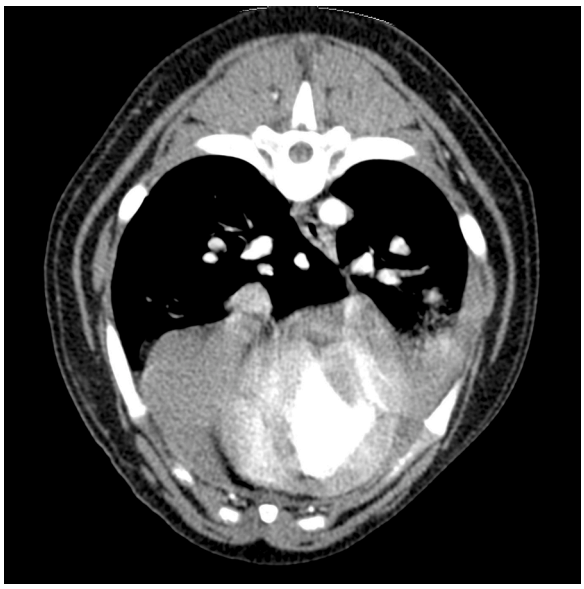

Image 1. Findings of computed tomography revealed residual pleural effusion, passive and isolated secondary lung atelectasis, soft tissue attenuating mass in the ventral mediastinum compatible with hematoma, severe thymic involution, sternal and mediastinal lymphadenomegaly.

and thoracic POCUS were unremarkable. Neurological examination identified severe cervical hyperesthesia, lateral recumbency, non-ambulatory tetraparesis, absent hindlimb postural reactions and absent spinal reflexes. All these findings were suggestive of a C6–T2 neurological lesion. Prior to magnetic resonance imaging (MRI) a complete blood cell count, biochemistry, and coagulation profile, three view thoracic radiographs and abdominal ultrasound exam were performed. All tests were unremarkable and subsequently an MRI of C6-T2 was performed. MRI results showed a marked and extensive hyperintense intramedullary lesion on T2, with thickening of the spinal cord and areas of "signal void" in the same sequence, compatible with bleeding. In the postcontrast sequence a marked meningeal uptake was detected. (Image 2). The cerebrospinal fluid analysis indicated an elevated C-reactive protein (CRP) level at 126.2 mg/L (normal range: 0 – 10.7). Cytology revealed polymorphonuclear pleocytosis with a significant presence of erythrocytes. These findings are indicative of a diagnosis consistent with steroid-responsive meningitis-arteritis (SRMA) accompanied by intramedullary hemorrhage. Prednisone was started at a dose of 4 mg/kg/day and the patient showed a dramatic clinical improvement over the next 24 hours of hospitalization. The patient was discharged 24 hours later under the care of her owners again with only prednisone as a treatment. Three weeks after diagnosis the dog was

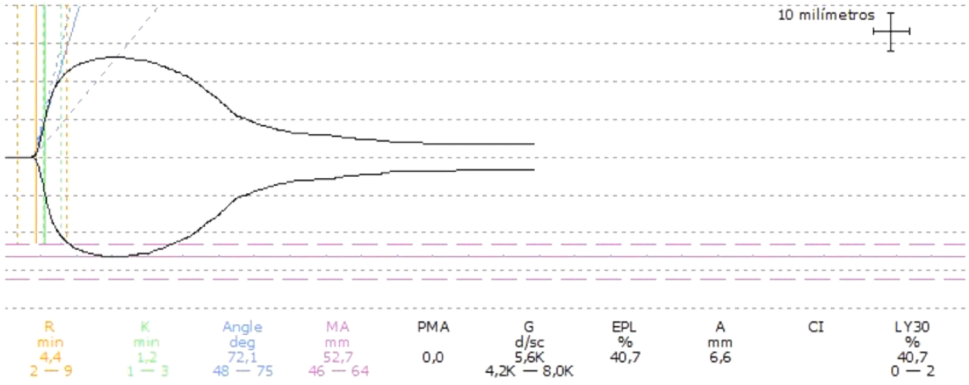

Figure 1. Results of thromboelastography TEG showed a remarkable hyperfibrinolytic pattern.

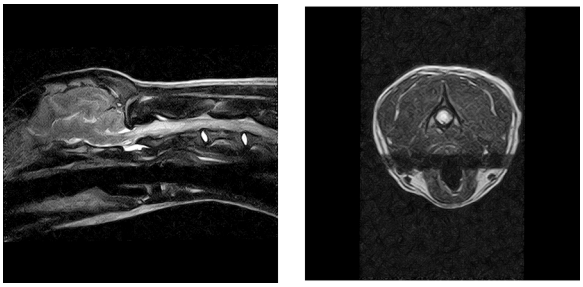

Image 2. Findings of magnetic resonance imaging. A. Sagittal section (left) of the brain and cervical spine TW2 shows diffuse intramedullary hyperintensity. B. Transverse section (right) Tw2 of the cervical spine shows intramedullary hyperintensity.

clinically normal. Prednisone was gradually tapered over the following months, guided by regular neurological and clinical examinations and CRP measurements, without any relapses.

Discussion

This report describes a case of a dog with spontaneous hemothorax due to thymic hemorrhage and its potential association to steroid-responsive meningitis-arteritis (SRMA) disease. Spontaneous hemothorax has been associated with either local factors (non-coagulopathic hemothorax) or secondary to systemic disorders affecting primary and/or secondary hemostasis.[6] The prevalence of non-coagulopathic spontaneous hemothorax in dogs is unknown. A variety of etiologies have been documented, with neoplasia being one of the most frequent etiologies.[2,7] Lung lobe torsion,[1] pulmonary thromboembolism,[8] diaphragmatic hernia,[1] pancreatitis,[2,3] infectious diseases,[2,3] spontaneous vascular rupture [1,9] and thymic hemorrhage/involution [1-3] have also been reported as the cause.

In our patient, we were initially able to identify thymic hemorrhage/involution as a potential explanation for the spontaneous hemothorax. Neoplasia was ruled out based on imaging findings and cytological analysis of pleural effusion. CT scan also helped to rule out other causes such as lung lobe torsion, pulmonary thromboembolism, vascular abnormalities, and diaphragmatic hernia. Pancreatitis was also ruled out based on CT scan and lack of clinical signs compatible with it.[10] However, a quantitative pancreatic lipase was never measured since this diagnosis was considered unlikely.

Infectious diseases associated with spontaneous non-coagulopathic hemothorax reported in the literature include spirocercosis [3], dirofilariasis [2,3,11], and streptococcosis [2,3]. Infection by *Dirofilaria immitis* was ruled out using a Snap 4Dx test. Spirocercosis was considered very unlikely due to its low prevalence in our country; however, no specific test was performed to rule it out. Nevertheless, the patient recovered well without specific treatment for spirocercosis. Blood tests for streptococcosis were not performed. Reported cases of *Streptococcus equi* bronchopneumonia are associated with hemothorax. [3] The absence of other clinical findings consistent with *Streptococcus equi* and improvement without antibiotic treatment makes this diagnosis a very unlikely cause of hemothorax.

Thymic hemorrhage is a reported unusual cause of spontaneous hemothorax in dogs. It has been associated with a wide variety of causes, including traumatic event to the thorax, excessive pulling on a dog's collar, rupture of aneurysm, underlying neoplasia such as thymoma or thymic lymphoma, ingestion of anticoagulant rodenticides, and even spontaneous thymic hemorrhage with no evident etiology.[4,12] The proposed underlying mechanisms for the development of fatal thymic hemorrhage in these idiopathic cases rely on the assumption that thymic involution was developing in affected dogs before the onset of hemorrhage.[13] Thymic involution is a physiologic process which occurs between 6 and 12 months of age in dogs.[4,6] Thymic involution is characterized by a gradual depletion of cortical lymphocytes, replacement of parenchyma by adipose tissue, and an increase in the number of thymic corpuscles. In addition, it is hypothesized that a regressing thymus containing a fragile, thin-walled vascular bed that lacks support from the adjacent fibroadipose tissue might be predisposed to fatal hemorrhage after a sudden increase in blood pressure or even after a minor traumatic event.[4,6] Although cytological or histopathological examination of thymic tissue could help in the diagnosis, the documented tissue regression and difficult access prevented the cytological diagnosis.[4]

Given our patient's pronounced hyperfibrinolysis on Thromboelastography (TEG), we postulated that an inflammatory condition, such as an emerging steroid-responsive meningitis-arteritis (SRMA), might have triggered a pro-inflammatory state. This, in turn, could have led to increased fibrinolysis and substantial thymic hemorrhage during the involution process. [14,15] The correlation between systemic inflammation and heightened fibrinolysis has been documented in humans.[16] Nevertheless, secondary hyperfibrinolysis in dogs has been reported in various disease processes, including disseminated intravascular coagulation (DIC), acute coagulopathy of trauma-shock (ACOTS), endothelial damage, and the release of catecholamines, often associated with hypoperfusion. Notably, hemothorax has been documented in cats.[15,16] In our case, the patient experienced hypoperfusion and catecholamine release due to hemorrhagic shock upon admission, leading to blood insufficiency and tissue hypoxia. This scenario could potentially explain the observed state of hyperfibrinolysis. Furthermore, endothelial damage within the thoracic cavity, attributed to thymic involution and hemorrhage, may have contributed to the observed hyperfibrinolysis.

Steroid-responsive meningitis-arteritis is an immune-mediated inflammatory disorder of the central nervous system (CNS), characterized by an abnormal immune response targeting the leptomeninges and associated vessels. [17,18] Therefore, systemic vasculitis and perivasculitis can develop and potentially interfere with the patient's hemostatic response.[17-19] In fact, increased fibrinolysis has been described in the cerebrospinal fluid of dogs with SRMA.[14] Although more studies are needed to know the aetiopathological mechanism of SRMA, there is increasing evidence that this disorder affects systemic vascular beds, organs outside of nervous system, such us arteries of the heart, cranial mediastinum, and cervical spinal meninges.[5,17] In the author's opinion, this could be the underlying mechanism that would explain our patient's initial presentation.

Other potential causes of a hyper fibrinolytic state include recent trauma with severe hypoperfusion, disseminated intravascular coagulation (DIC), cavitary effusions, breed predisposition and infection by *Angiostrongylus vasorum*.[20] Trauma was considered unlikely in this dog based on the history. DIC with enhanced hyperfibrinolysis could explain this patient presentation. The diagnosis of this type of DIC is made using the ratio of fibrin degradation products (FDP's) and d-dimers, elevation of thrombin-antithrombin (TAT), plasmin-α2 plasmin inhibitor complex (PIC), fibrinogen $>$ 100 mg/dl, and platelet count $>$ 50.000 u/L. Although FDP's, PIC, TAT and d-dimers were not measured the fact that our patient presented a platelet count and fibrinogen within the reference range make this diagnosis unlikely.

While other breeds, such as the greyhound, have been described as to demonstrate a hyper fibrinolytic basal status, to the author's knowledge, this has not been reported in golden retrievers, making this potential association unlikely. Infection due to *Angiostrongylus vasorum* was also ruled out using an in-house snap test, and serology was submitted, which yielded a negative result. The presence of cavitary effusions can lead to systemic hyperfibrinolysis. The mechanism behind this is thought to occur because of resorption of hyperfibrinolytic fluid from the lymphatic circulation and subsequent return to the systemic circulation via the thoracic duct.[21] We cannot exclude this mechanism as the explanation for the marked hyperfibrinolysis in our patient.

Upon the initial presentation, our patient showed light neck pain, but at that time, respiratory distress was more evident. The work-up was

directed to determine the underlying cause of the respiratory distress. Prednisone was started early after major infectious causes were ruled out, to help decrease the intrathoracic inflammatory reaction observed along with tranexamic acid. At the time of the first discharge, we could not identify an underlying disease beyond spontaneous hemorrhage due to thymic involution that would explain the spontaneous hemothorax. The dog was continued on prednisone for over a week and then was gradually tapered. This likely masked the signs of an overt SMRA, which fully developed after the steroids were completely discontinued.

Upon the manifestation of overt neurological signs, the definitive diagnosis of SMRA was established by considering the patient's clinical symptoms, the evolution of symptoms over time, common dog breed, cerebrospinal fluid analysis encompassing cytology and CRP, and the conclusive identification of severe intramedullary bleeding through MRI.[18,22,23] Notably, no other instances of bleeding were observed at that point, and the coagulation profile appeared unremarkable. It's important to note that a TEG was not conducted on this occasion due to the limitation that our laboratory does not offer this technique outside regular operating hours.

Therefore, we cannot be certain if the patient was hyper fibrinolytic in this episode. Itś important to mention the intramedullary bleeding is not common in cases of SRMA, however vasculitis is more common in the leptomeninges of the spinal cord and in the chronic form of SRMA the nerve root degeneration and spinal cord infarction, secondary to rupture and hemorrhage of structurally weakened vessels may be present. Also, responsiveness to prednisone and a short course of tranexamic acid treatment with positive and quick response supports the diagnosis.

In conclusion, although further understanding of the underlying pathophysiology of SRMA and the development of spontaneous bleeding in areas outside of the CNS is needed, we believe that the dog presented in this report likely developed a hemostatic imbalance triggered by a neurological inflammatory disease, resulting in excessive hemorrhage during a physiological process such as thymic involution. To the best of the author's knowledge this is the first case report to describe such an unusual association.

CRediT authorship contribution statement

B Alcocer: Conceptualization, Visualization, Writing – original draft. **P Bou:** Visualization, Writing – review & editing. **L Bosch:** Visualization, Writing – review & editing. **C Torrente:** Visualization, Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no conflict of interest.

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