Acta Scientiae Veterinariae, 2026. 54(Suppl 1): 1114.

CASE REPORT
Pub. 1114

ISSN 1679-9216

Mediastinal Lymphoma in a Bitch: Clinical Management of Thrombotic Complications

Junghun Oh, Seonggyu Jeong & Joonghyun Song

ABSTRACT

***Background*:** The association between neoplasia and hypercoagulability has received increasing recognition, and lymphoma is one of the neoplastic diseases associated with thromboembolic complications. In human medicine, reduced mobility, tumor location, and chemotherapeutic agents are recognized as established risk factors for thrombosis. This report describes one bitch with T-cell mediastinal lymphoma complicated by pulmonary thromboembolism (PTE) and iliac vein thrombosis, emphasizing the importance of recognizing thrombosis-related risk factors in canine lymphoma.

***Case*:** A 10-year-old spayed bitch Tosa was presented with vomiting, diarrhea, and anorexia. Computed tomography (CT) revealed a cranial mediastinal mass. Fine-needle aspiration cytology and polymerase chain reaction for antigen receptor rearrangement confirmed a diagnosis of high-grade T-cell mediastinal lymphoma. Prednisolone monotherapy was initiated. Three months later, the patient was re-presented with respiratory distress and right hindlimb swelling. Thromboelastography (TEG) showed hypercoagulability. Portable glucose and lactate measurements revealed decreased glucose and increased lactate in the right hindlimb relative to the left, suggesting an ischemic condition. Thoracic radiographs demonstrated a moderate volume of pleural effusion. CT revealed concurrent PTE and right iliac vein thrombosis, along with enlargement of the mediastinal mass and regional lymph nodes compressing the cranial vena cava. Treatment with L-asparaginase and lomustine was initiated, and antithrombotic agents including clopidogrel and rivaroxaban were concurrently administered. Three weeks after the treatment initiation, blood glucose levels and lactate concentrations in right hindlimb returned to normal levels. TEG showed an improvement in hypercoagulability. Thoracic radiographs showed resolution of pleural effusion. These findings indicated that thrombotic complications were successfully controlled. However, the patient died 4 months after initial diagnosis of mediastinal lymphoma.

***Discussion*:** This report describes a case of pulmonary thromboembolism and iliac vein thrombosis in a bitch with mediastinal lymphoma, suggesting potential risk factors that may have contributed to thrombogenesis. Mediastinal lymphoma, as demonstrated in this animal, can mechanically compress major vessels such as the cranial vena cava, predisposing to venous stasis and thrombus formation. This mechanism is consistent with observations in human medicine, where vascular compression by mediastinal tumors markedly increases thrombotic risk. Chemotherapeutic agents such as glucocorticoids and L-asparaginase are known to decrease antithrombin synthesis and enhance platelet activity, thereby amplifying thrombotic potential in patients already predisposed by neoplasia. Furthermore, reduced mobility related to lethargy or hospitalization can intensify venous stasis, a mechanism well established in human oncology. These interacting factors likely acted synergistically in this patient to promote thrombus formation. TEG proved valuable for monitoring dynamic coagulation changes, sensitively reflecting both the hypercoagulable state and subsequent improvement with treatment. Incorporating such monitoring may help clinicians detect early coagulation imbalance and evaluate therapeutic response in real time. Overall, this case highlights that in canine lymphoma, vascular compression within the mediastinal space, the use of chemotherapeutic agents, and patient immobility can collectively increase the risk of thrombosis. Recognizing these concurrent risk factors is crucial for timely intervention. TEG-based monitoring and early intervention may improve clinical outcomes in dogs with mediastinal lymphoma.

Keywords: dog, canine, haematopoietic neoplasia, mediastinal lymphoma, thromboelastography, thromboembolism.

DOI: 10.22456/1679-9216.151528

Received: 20 January 2026 Accepted: 13 July 2026 Published: 7 August 2026

Department of Veterinary Internal Medicine, College of Veterinary Medicine, Chungnam National University (CNU), Daejeon, South Korea. CORRESPONDENCE: J. Song [jh.song@cnu.ac.kr]. Department of Veterinary Internal Medicine, College of Veterinary Medicine, Chungnam National University (CNU). Daejeon 34134, South Korea.

INTRODUCTION

Lymphoma is the most common haematopoietic neoplasia in the dog [4]. Mediastinal lymphoma accounts for approximately 5% of canine lymphoma that is almost exclusively of T-cell origin [20]. Major complications of mediastinal lymphoma include vena cava syndrome, in which the tumor compresses the cranial vena cava (CrVC) causing head and forelimb edema, and respiratory signs such as dyspnea or cough due to mass effect or pleural effusion [20].

In veterinary medicine, the relationship between neoplasia and hypercoagulability has received increasing attention [3]. Retrospective studies have identified neoplasia as the most common concurrent condition associated with thrombosis, accounting for 41% of dogs with pulmonary thromboembolism (PTE) and 54% of dogs with splenic vein thrombosis (SVT) [9,13]. Lymphoma featured prominently among the neoplasms identified, accounting for 42% of PTE cases and 21% of SVT cases [9,13]. In addition, prospective study demonstrated that up to 81% of dogs with multicentric lymphoma exhibit laboratory evidence of hypercoagulability at diagnosis [12].

Similarly, in human medicine, lymphoma is recognized as a high-risk neoplasm for thrombosis [10]. A meta-analysis reported a 6.5% incidence of thrombosis in human patients with non-Hodgkin's lymphoma [2]. Risk factors for thromboembolism (TE) include patient variables such as age, obesity, and immobility, and disease-specific factors like tumor site and chemotherapy [8].

This case report describes the clinical presentation, diagnostic evaluation, therapeutic management, and outcome of a bitch with T-cell mediastinal lymphoma complicated by concurrent PTE and iliac vein thrombosis.

CASE

A 10-year-old spayed bitch Tosa presented with vomiting, diarrhea, and anorexia. Thoracic radiographs showed soft tissue opacity in the cranial mediastinum. On computed tomography (CT), a well-defined mass measuring 10.0×5.6×5.0 cm was identified in the cranial mediastinum (Figure 1A-B). Ultrasound-guided fine-needle aspiration of the mediastinal mass was performed. Cytological examination revealed that there were more than 50% intermediate-to-large lymphoid cells, suggesting high grade lymphoma (Figure 2). Clonality was confirmed by polymerase chain reaction for antigen receptor rearrangement (PARR), which demonstrated a monoclonal T-cell receptor gene rearrangement [Canine Lymphoma PCR Test][1]. Based on cytological examination and PARR, the mediastinal mass was diagnosed as high-grade T-cell lymphoma. Due to financial constraints of the caregiver, prednisolone[2] [50 mg/m² orally every 24 h] monotherapy was administered as palliative treatment.

Three months later, the patient was re-presented with respiratory distress and swelling of the right hindlimb. Physical examination revealed lethargy, rapid breathing, and generalized enlargement of peripheral lymph nodes. A non-painful, non-pitting edema was present in the right hindlimb extending distally from the thigh to the digits (Figure 3A). Blood glucose concentrations, which were measured using a portable glucometer [VetMate®][3] from each hindlimb, showed different values between the left and right sides (left vs. right, 6.8 vs. 3.2 mmol/L). Concurrently, blood lactate concentrations, measured using a portable lactate analyzer [StatStrip Xpress Lactate®][4], also showed different values between the hindlimbs (left vs. right, 2.4 vs. 7.3 mmol/L). These findings were suggestive of right hindlimb ischemia. D-dimer levels were increased (0.5 mg/L; reference interval [RI]: 0-0.3 mg/L), as measured using a fluorescence immunoassay analyzer [Vcheck V200®]5. Kaolin-activated Thromboelastography (TEG) analysis [Haemoscope TEG® 5000][6] revealed hypercoagulable state as follows: reaction time (R) = 1.2 min (RI: 1.8-8.6 min), clot formation time (K) = 0.8 min (RI: 1.2-3.9 min), angle (α) = 80.5° (RI: 45.5-73.5°), maximum amplitude (MA) = 74.9 mm (RI: 46.8-66.1 mm). Thoracic radiography revealed an enlarged cranial mediastinal mass and pleural effusion (Figure 4A). Fluid obtained by thoracentesis was characterized as a modified transudate. These findings were suggestive of suspected thrombosis in the right iliac or femoral vein and the pulmonary vessels. CT confirmed the presence of thrombosis, revealing intraluminal filling defects within the pulmonary arteries and the right internal iliac vein, consistent with PTE and iliac vein thrombosis (Figure 5A-B). CT further revealed that the cranial mediastinal mass had increased in size to 11.8×5.9×5.3 cm, and concurrent enlargement of surrounding mediastinal lymph nodes, resulting in compression of the CrVC (Figure 5C-D). After establishing the diagnosis of PTE and

iliac vein thrombosis, clopidogrel bisulfate[7] [10 mg/kg orally loading dose, then 3 mg/kg orally every 24 h] and rivaroxaban[8] [Xarelto®; 2 mg/kg orally every 24 h] were administered. The chemotherapy protocol was modified to include L-asparaginase[9] [400 U/kg, subcutaneously every 21days], lomustine[10] [60 mg/m² orally every 21 days] and the dose of prednisolone [2 mg/kg orally every 24 h] was also adjusted. One week after the initiation of treatment, a visible reduction in right hindlimb swelling was noted (Figure 3B). Blood glucose levels (left vs. right: 5.9 vs. 6.6 mmol/L) and lactate concentrations (left vs. right: 2.8 vs. 2.9 mmol/L) demonstrated comparable values between both limbs, indicating recovery from ischemia in the right hindlimb. Thoracic radiography revealed stable disease, with no significant change in the size of the mediastinal mass and a slight decrease in the volume of pleural effusion (Figure 4B).

Three weeks after the initiation of treatment for thrombosis, the right hindlimb had returned to normal appearance (Figure 3C). TEG showed an improvement in hypercoagulability as follows: R = 3.9, K = 1.1, α = 75.1, MA = 76.9. In addition, thoracic radiography revealed resolution of pleural effusion (Figure 4C), and a slight reduction in the size of the mediastinal mass. These findings indicated that thrombotic complications were successfully controlled. Despite the chemotherapy, the lymphoma did not achieve remission and remained uncontrolled, and the patient died 4 months after the initial diagnosis.

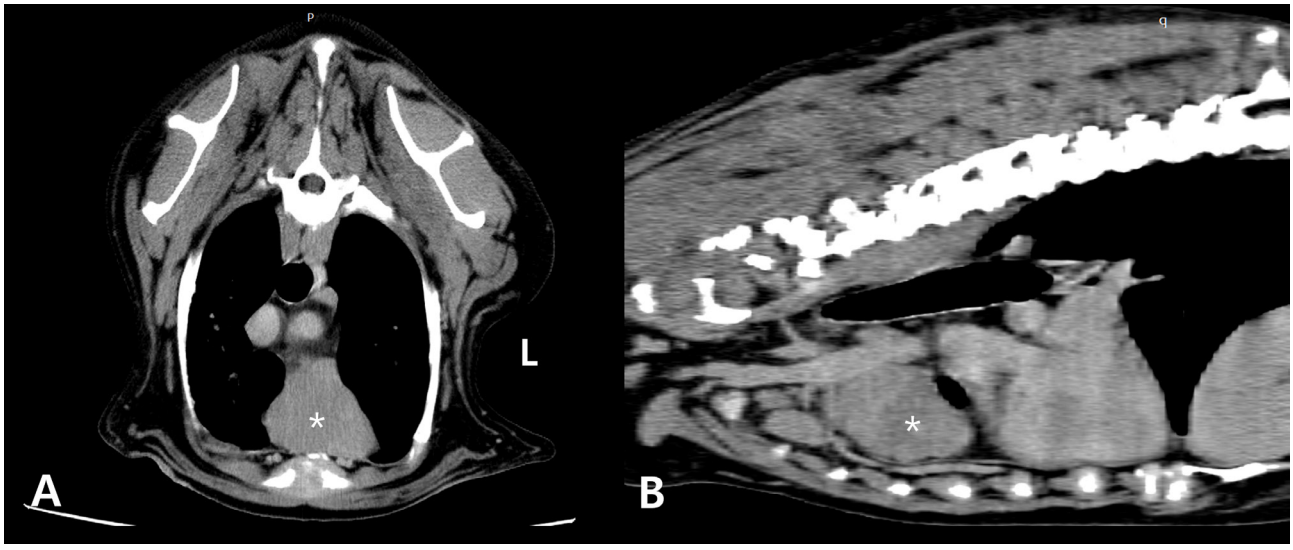

Figure 1. A- Transverse & B- Sagittal-plane computed tomography (CT) images from the initial presentation. A & B- CT images revealed a large, well-demarcated soft tissue mass (asterisk) occupying the cranial thoracic cavity.

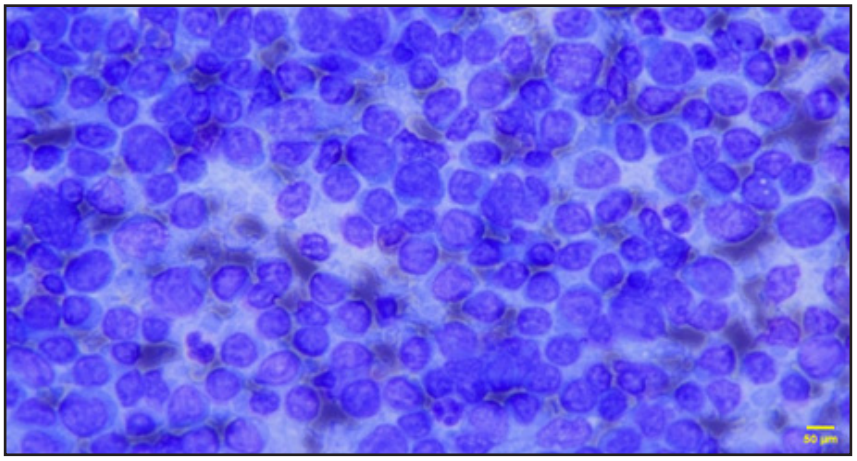

Figure 2. Fine-needle aspiration (FNA) cytology of the mediastinal mass. Cytologic analysis revealed a predominance of intermediate-to-large lymphoid cells, consistent with high grade lymphoma [Diff-Quik stain, ×400; Scale bar= 20 µm].

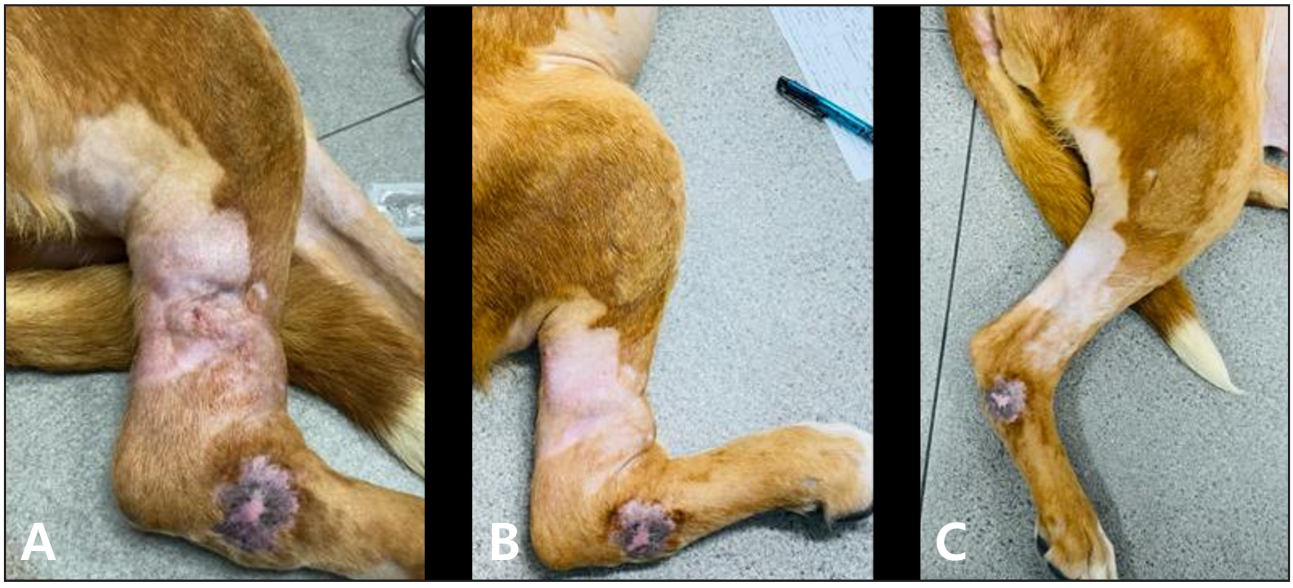

Figure 3. Clinical course of right hindlimb edema in a dog with iliac vein thrombosis following antithrombotic therapy. A- Marked pitting edema extending from the thigh to the digits was observed at the time of re-presentation. B- One week after treatment, the limb showed visible reduction in swelling. C- By 3 weeks after treatment, the right hindlimb had returned to near-normal size, with resolution of edema.

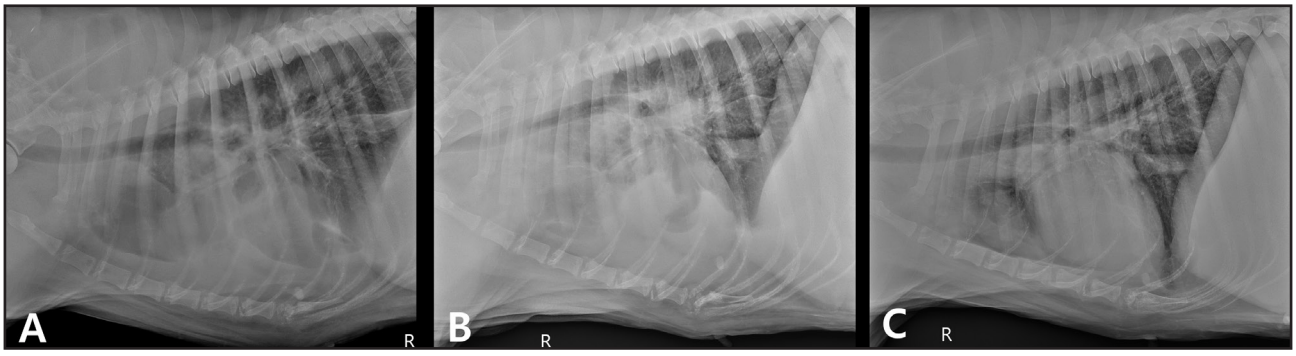

Figure 4. Right lateral (A-C) thoracic radiographs from the present case. A- Radiograph obtained at re-presentation shows pleural effusion. B- Radiograph obtained 1 week after initiation of treatment shows mild improvement of pleural effusion. C- Radiograph obtained 3 weeks after treatment demonstrates marked resolution of the pleural effusion.

DISCUSSION

Mediastinal lymphoma is a well-recognized risk factor for thrombosis in human patients [8]. In human studies, mediastinal lymphoma patients show a TE incidence of 35.7%, higher than the overall 6.4% reported in lymphoma patients [2,14]. This increased risk is thought to reflect the anatomical feature of the mediastinum, where expanding tumors can compress major vessels. Human studies have reported that patients with mediastinal lymphoma are at increased risk of thrombosis due to direct vascular compression [6,14]. This compression promotes venous stasis, local hypoxia, endothelial activation, and inflammation [1]. In the present case, compression of the CrVC caused by mediastinal lymph node enlargement was observed, accompanied by the development of thrombus formation. Such mechanical compression of a major vessel likely increased the risk of thrombosis by causing venous stasis, thereby exacerbating the hypercoagulable state. Therefore, mediastinal lymphoma should be recognized in veterinary medicine as a condition with a high thrombotic risk, particularly when vascular compression is evident.

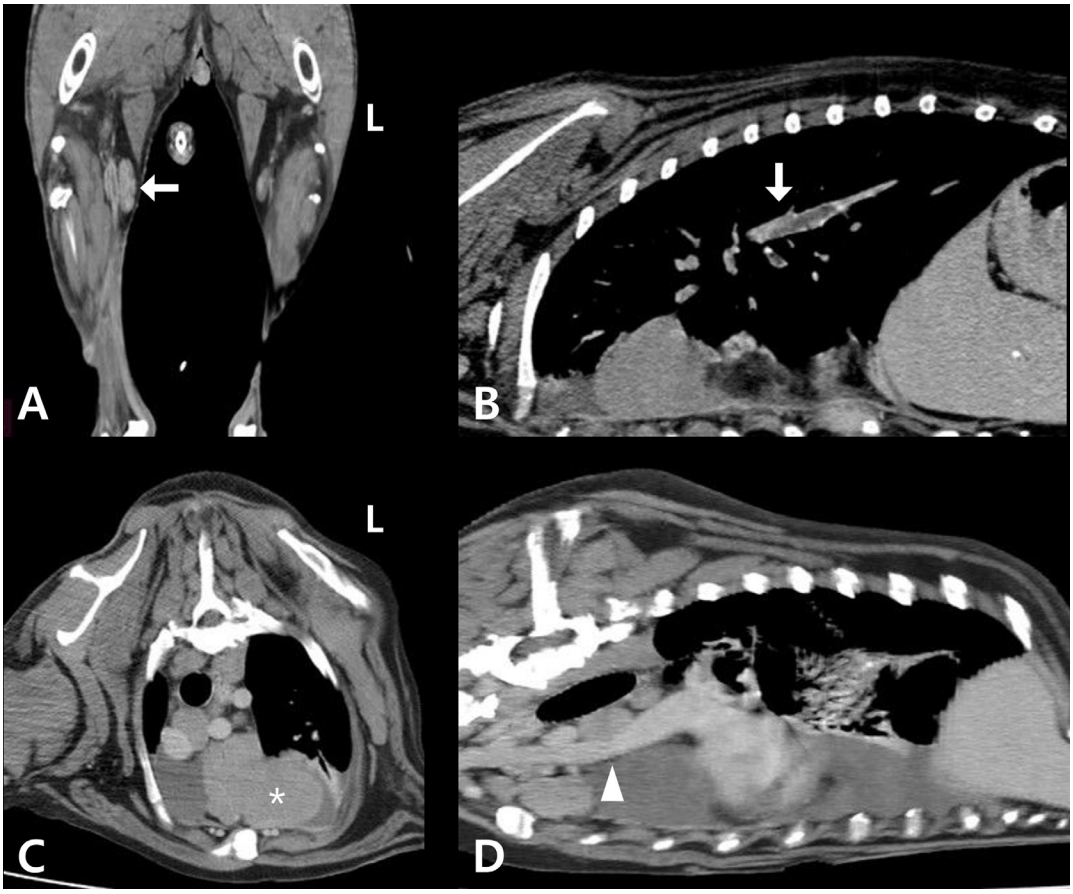

Figure 5. Computed tomography (CT) multiplanar reconstruction (MPR) images obtained at the time of re-presentation. A- Coronal-plane CT image reveals a well-defined, oval shaped filling defect (arrow) with soft tissue attenuation within the lumen of the right internal iliac vein. B- Sagittal-plane CT image reveals multiple irregular filling defects (arrow) with soft tissue attenuation in the pulmonary arteries, consistent with pulmonary thromboembolism. C & D- Transverse and sagittal-plane CT images reveal an increase in the size of the cranial mediastinal mass (asterisk), along with compression of the cranial vena cava (arrowhead).

Chemotherapeutic agents such as glucocorticoids and L-asparaginase may further exacerbate hypercoagulability in dogs with lymphoma [16-18]. Prednisone induces hypercoagulability by reducing hepatic synthesis of antithrombin and enhancing platelet aggregation, whereas L-asparaginase promotes a hypercoagulable state by decreasing antithrombin III and suppressing hepatic protein synthesis [16-18]. Other agents, such as vincristine and doxorubicin, are also known to increase thrombotic risk in human medicine; doxorubicin can cause endothelial injury and disrupt the protein C pathway, whereas vincristine may induce vascular damage and platelet activation [7]. In the present case, the administration of glucocorticoids and L-asparaginase may have further promoted thrombus formation in a patient already predisposed to hypercoagulability due to lymphoma. These findings indicate that chemotherapeutic agents commonly used in the treatment of canine lymphoma, such as glucocorticoids, L-asparaginase, vincristine, and doxorubicin, may contribute to therapy-associated hypercoagulability. This highlights the importance of close monitoring for thromboembolic complications throughout the course of treatment.

Immobility caused by poor performance status, hospitalization, or recent surgery is also a recognized risk factor for TE in human lymphoma patients [5,19]. In veterinary medicine, the relationship between physical activity and hypercoagulability is not well established. However, critically ill dogs often exhibit reduced mobility due to lethargy, particularly during hospitalization, which may be associated with an increased risk of TE through mechanisms similar to human medicine. In the

present case, lethargy and reduced mobility were observed and may have further increased the risk of thrombosis associated with mediastinal lymphoma. The benefits of early mobilization for thrombosis prevention are well-documented in humans [15]. Therefore, encouraging mobility and promoting preventive physical activity should be considered in dogs with conditions predisposing to thrombosis, such as mediastinal lymphoma.

In dogs with lymphoma, careful monitoring for thromboembolic complications is essential during treatment, as factors such as chemotherapeutic agents and immobility can further promote hypercoagulability. TEG may serve as a valuable tool for this purpose, as it provides a comprehensive evaluation of the entire coagulation process and is highly sensitive in detecting hypercoagulable states [11]. In this case, thrombus formation was accompanied by significant TEG changes consistent with a hypercoagulable state. These abnormalities subsequently resolved concurrently with clinical recovery. These observations demonstrate that TEG can sensitively reflect dynamic changes in hypercoagulability associated with neoplastic diseases such as lymphoma. Therefore, in dogs with additional thrombotic risk factors, such as mediastinal lymphoma, reduced mobility, or chemotherapy, TEG-based monitoring and risk assessment may play a valuable role in predicting thrombus formation and monitoring therapeutic response.

In conclusion, this report describes a case of mediastinal lymphoma in a bitch complicated by TE. Mediastinal lymphoma poses a particularly high thrombotic risk due to vascular compression within the mediastinum. Moreover, factors such as chemotherapy and reduced mobility may further exacerbate hypercoagulability in oncology patients. These findings highlight the importance of vigilant, real-time monitoring of coagulation status in dogs at risk for thrombosis. TEG may serve as a valuable adjunct for detecting hypercoagulable states and assessing therapeutic response in such cases.

MANUFACTURERS

[1]Korea Vet Lab Inc. Seoul, South Korea.

[2]Yuhan Co. Seoul, South Korea.

[3]i-SENS Inc. Seoul, South Korea.

[4]Nova Biomedical. Waltham, MA, USA.

[5]Bionote Co. Ltd. Hwaseong, South Korea.

[6]Haemonetics Corp. Boston, MA, USA.

[7]Samjin Pharmaceutical Co. Ltd. Seoul, South Korea.

[8]Bayer HealthCare Pharmaceuticals Inc. Whippany, NJ, USA.

[9]Kyowa Hakko Kirin Co. Ltd., Shizuoka, Japan.

[10]Medac GmbH. Wedel, Germany.

***Funding*.** This study was supported by the Basic Science Research Program through the NRF funded by the Ministry of Education (RS-2023-00219710314820926400001).

***Declaration of interest*.** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

1 **Bovill E.G. & van der Vliet A. 2011.** Venous valvular stasis–associated hypoxia and thrombosis: what is the link? *Annual Review of Physiology*. 73(1): 527-545. DOI: 10.1146/annurev-physiol-012110-142305

2 **Caruso V., Di Castelnuovo A., Meschengieser S., Lazzari M.A., De Gaetano G., Storti S., Iacoviello L. & Donati M.B. 2010.** Thrombotic complications in adult patients with lymphoma: a meta-analysis of 29 independent cohorts including 18 018 patients and 1149 events. *Blood, The Journal of the American Society of Hematology*. 115(26): 5322-5328. DOI: 10.1182/blood-2010-01-258624

3 **De Laforcade A. 2012.** Diseases associated with thrombosis. *Topics in companion animal medicine*. 27(2): 59-64. DOI: 10.1053/j.tcam.2012.07.002

4 **Dobson J., Samuel S., Milstein H., Rogers K. & Wood J. 2002.** Canine neoplasia in the UK: estimates of incidence rates from a population of insured dogs. *Journal of Small Animal Practice*. 43(6): 240-246. DOI: 10.1111/j.1748-5827.2002.tb00066.x

5 **Engbers M., Blom J., Cushman M., Rosendaal F. & van Hylckama Vlieg A. 2014.** The contribution of immobility risk factors to the incidence of venous thrombosis in an older population. *Journal of Thrombosis and Haemostasis*. 12(3): 290-296. DOI: 10.1111/jth.12480

6 **Gartrell J., Kaste S.C., Sandlund J.T., Flerlage J., Zhou Y., Cheng C., Estepp J. & Metzger M.L. 2020.** The association of mediastinal mass in the formation of thrombi in pediatric patients with non-lymphoblastic lymphomas. *Pediatric Blood & Cancer*. 67(2): e28057. DOI: 10.1002/pbc.28057

7 **Grover S.P., Hisada Y.M., Kasthuri R.S., Reeves B.N. & Mackman N. 2021.** Cancer therapy–associated thrombosis. *Arteriosclerosis, Thrombosis, and Vascular Biology*. 41(4): 1291-1305. DOI: 10.1161/ATVBAHA.120.314378

8 **Hohaus S., Bartolomei F., Cuccaro A., Maiolo E., Alma E., D'Alò F., Bellesi S., Rossi E. & De Stefano V. 2020.** Venous thromboembolism in lymphoma: risk stratification and antithrombotic prophylaxis. *Cancers*. 12(5): 1291. DOI: 10.3390/cancers12051291

9 **Johnson L.R., Lappin M.R. & Baker D.C. 1999.** Pulmonary thromboembolism in 29 dogs: 1985–1995. *Journal of Veterinary Internal Medicine*. 13(4): 338-345. DOI: 10.1111/j.1939-1676.1999.tb02192.x

10 **Khorana A.A., Kuderer N.M., Culakova E., Lyman G.H. & Francis C.W. 2008.** Development and validation of a predictive model for chemotherapy-associated thrombosis. *Blood, The Journal of the American Society of Hematology*. 111(10): 4902-4907. DOI: 10.1182/blood-2007-10-116327

11 **Kol A. & Borjesson D.L. 2010.** Application of thrombelastography/thromboelastometry to veterinary medicine. *Veterinary Clinical Pathology*. 39(4): 405-416. DOI: 10.1111/j.1939-165X.2010.00263.x

12 **Kol A., Marks S., Skorupski K., Kass P., Guerrero T., Gosselin R. & Borjesson D. 2015.** Serial haemostatic monitoring of dogs with multicentric lymphoma. *Veterinary and Comparative Oncology*. 13(3): 255-266. DOI: 10.1111/vco.12041

13 **Laurenson M., Hopper K., Herrera M. & Johnson E. 2010.** Concurrent diseases and conditions in dogs with splenic vein thrombosis. *Journal of veterinary internal medicine.* 24(6): 1298-1304. DOI: 10.1111/j.1939-1676.2010.0593.x

14 **Lekovic D., Miljic P. & Mihaljevic B. 2010.** Increased risk of venous thromboembolism in patients with primary mediastinal large B-cell lymphoma. *Thrombosis Research*. 126(6): 477-480. DOI: 10.1016/j.thromres.2010.08.017

15 **Raya-Benítez J., Heredia-Ciuró A., Calvache-Mateo A., Martín-Núñez J., Valenza-Peña G., López-López L. & Valenza M.C. 2025.** Effectiveness of non-instrumental early mobilization to reduce the incidence of deep vein thrombosis in hospitalized patients: A systematic review and meta-analysis. *International Journal of Nursing Studies*. 161: 104917. DOI: 10.1016/j.ijnurstu.2024.104917

16 **Rogers K.S., Barton C.L., Benson P.A. & Green R.A. 1992.** Effects of single-dose L-asparaginase on coagulation values in healthy dogs and dogs with lymphoma. *American Journal of Veterinary Research*. 53(4): 580-584. DOI: 10.2460/ajvr.1991.53.04.580

17 **Romão F.G., Campos E.F., Mattoso C.R.S. & Takahira R.K. 2013.** Hemostatic profile and thromboembolic risk in healthy dogs treated with prednisone: a randomized controlled trial. *BMC Veterinary Research*. 9(1): 268. DOI: 10.1186/1746-6148-9-268

18 **Rose L.J., Dunn M.E., Allegret V. & Bédard C. 2011.** Effect of prednisone administration on coagulation variables in healthy B eagle dogs. *Veterinary Clinical Pathology*. 40(4): 426-434. DOI: 10.1111/j.1939-165X.2011.00364.x

19 **Trujillo-Santos J., Gussoni G., Gadelha T., Muñoz-Torrero J.F.S., Barron M., Lopez L., Ruiz-Ruiz J., Fernandez-Capitan C., Lorente M. & Monreal M. 2014.** Influence of recent immobilization or surgery on mortality in cancer patients with venous thromboembolism. *Thrombosis research*. 133: S29-S34. DOI: 10.1016/S0049-3848(14)50005-9

20 **Zandvliet M. 2016.** Canine lymphoma: a review. *Veterinary Quarterly*. 36(2): 76-104. DOI: 10.1080/01652176.2016.1152633