Acta Scientiae Veterinariae, 2026. 54(Suppl 1): 1103.

CASE REPORT
Pub. 1103

ISSN 1679-9216

Esophageal Leiomyoma-Associated Angiodysplasia in a Dog: A Long-Term Management Strategy

Hyunjin Kim & Joonghyun Song

ABSTRACT

***Background*:** Angiodysplasia [AGD] is an uncommonvascular malformation defined as abnormally dilated and tortuous vessels within mucosal and submucosal layers, representing a rare cause of gastrointestinal [GI] bleeding in veterinary medicine. While AGD typically occurs inthe lower GI tract, its occurrence in the esophagus has not been previously reported in dogs. This report presentsthe first canine case of esophageal AGD, which was found to be associated with an esophageal leiomyoma at the gastroesophageal junction [GEJ]. It describes the clinicopathologic findings, proposes a pathophysiologic mechanism linking mechanical venous obstruction to vascular ectasia, and presents a long-term, minimally invasive management strategy.

***Case*:** A 6-year-old neutered male mongrel dog was presented with a 3-month history of lethargy and anemia. Hematologic evaluation revealed severe microcytic, hypochromic anemia with markedly decreased serum iron concentration and transferrin saturation, consistent with iron deficiency anemia. Endoscopy identified dilated and tortuous vessels at the GEJ, and computed tomography confirmed a well-defined extraluminal esophageal mass compressing the distal esophagus. Histopathologic and immunohistochemical examinations confirmed an esophageal leiomyoma. Chronic extraluminal venous compression by the leiomyoma was suspected to contribute to localized venous stasis and secondary mucosal vascular ectasia. The dog was treated with oral ethinyl estradiol and levonorgestrel, combined with surgical debulking because complete resection was not feasible due to the mass location. Anemia resolved within 10 days of initiating hormonal therapy and did not recurduring 93 weeks of continuous hormonal treatment. At 53 weeks after the initiation of hormonal therapy, an intraluminal recurrence of the leiomyoma was detected and endoscopically removed; however, no recurrence of AGD or anemia was observed. Hormonal therapy was continued without adverse effects throughout follow-up.

***Discussion*:** This case proposes a novel pathophysiologic mechanism in which chronic venous outflow obstruction at the high-tension GEJ region [Laplace's law] may induce mucosal vascular ectasia consistent with AGD. Although a direct causal link cannot be definitively proven, the close anatomical association and sustained improvement of vascular lesions following decompression suggest that AGD developed secondary to venous obstruction caused by the leiomyoma. The findings extend the anatomic spectrum and pathophysiologic understanding of AGD in dogs. This case also underscores the diagnostic importance of integrating endoscopic and imaging modalities to identify underrecognized vascular lesions in anemic patients. Clinically, combined estrogen-progestin therapy, complemented by surgical debulking, achieved durable 93-week remission without recurrence of AGD, offering a feasible, minimally invasive option when invasive endoscopic ablation or surgery poses high-risk. Estrogen-progestin therapy likely promotes vascular stabilization through endothelial modulation and anti-angiogenic effects, providing durable hemostatic control. The absence of treatment-related complications supports the safety and efficacy of this long-term multimodal approach. This report broadens current knowledge of AGD in veterinary medicine and highlights a practical management strategy for tumor-associated AGD in anatomically constrained regions.

Keywords: angiodysplasia, dog, leiomyoma, hormone therapy, iron deficiency anemia.

DOI: 10.22456/1679-9216.151307

Received: 28 December 2025 Accepted: 27 May 2026 Published: 25 June 2026

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

INTRODUCTION

Angiodysplasia (AGD), also known as vascular ectasia, is characterized by abnormally dilated and tortuous blood vessels within the mucosal and submucosal layers, most commonly located in lower gastrointestinal [GI] tract [4,15]. Haemorrhage occurs when these fragile vessels rupture, leading to intermittent hematochezia or melena [3,6]. Diagnosis relies primarily on its macroscopic identificationof characteristic vascular lesions by endoscopy. The pathogenesis of AGD remains incompletely defined, while the leading hypothesis attributes lesion formation to chronic, low-grade venous obstruction [15]. In veterinary medicine, AGD is generally regarded as idiopathic, whereasit is usually associated with systemic diseases [e.g., aortic stenosis, chronic kidney disease, or von Willebrand disease] in humans [15]. AGD has also occasionally reported in association with neoplastic lesions; however, a direct causal link has not been conclusively established [16].

Endoscopic ablation is the mainstay of AGD treatment in humans [15]. When ablation fails, lesions are diffuse or inaccessible, or procedural risk is high, medical therapyis considered [15]. Surgical resection is reservedfor uncontrolled acute bleeding [15]. Only a limited number of canine cases have been reported, with management including surgical resection, endoscopic ablation, and hormonal therapy [2,4,14]. The prognosis is generally favourable in people, but long-term outcomesin canine patients remain poorly characterized due to the scarcity of reports. This report describes a case of esophageal AGD secondary to an esophageal leiomyoma in a dog, highlighting the diagnostic process and successful long-term management.

CASE

A 6-year-old neutered male mongrel dog was referred with a 3-month history of lethargy and anemia. Prior to referral, the dog had received prednisolone[1] [1.5 mg/kg - orally every 12 h], mycophenolate mofetil[2] [13 mg/kg - orally every 12 h], danazol[3] [5 mg/kg - orally every 12 h], iron dextran[4] [0.2 mL - intramuscularly] and blood transfusion. Despite these treatments, there was no clinical improvement, and both prednisolone and mycophenolate mofetil were gradually weaned off.

On presentation, physical examination revealed pale mucous membranes. Complete blood count [ProCyte Dx][5] identified microcytic [mean corpuscular volume (MCV) 52.4 fL; Ref.: 61.6-73.5], hypochromic [mean corpuscular hemoglobin concentration (MCHC) 267 g/L; Ref.: 320-379], severe anemia [hematocrit (HCT) 15%; Ref.: 37.3-61.7] with regeneration [reticulocyte count 238 × 10^9/L; Ref.: >110 × 10^9/L]. Manual packed cell volume was 20%. Leukogram abnormalities included neutrophilia and monocytosis, with hypersegmented neutrophils, consistent with a steroid leukogram. Platelet count was within the reference interval. Red blood cell morphology revealed numerous leptocytes and codocytes (Figure 1). These hematologic abnormalities were suggestive of iron deficiency anemia. Serum iron analysis[6] confirmed iron deficiency anemia, with decreased serum iron concentration [4.3 µmol/L; Ref.: 5.4-32.2] and transferrin saturation [4.55%; Ref.: 27-66], and normal total iron-binding capacity [94.3 µmol/L; Ref.: 50.8-101.9].

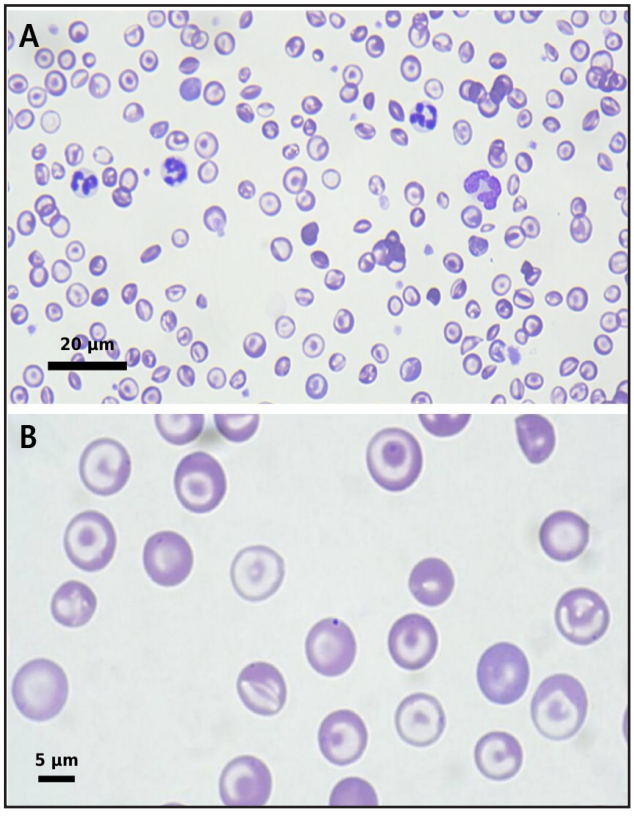

Figure 1. Peripheral blood smears. A- Microcytic and hypochromic red blood cells are observed [Diff-Quick stain ×400; Scale bar = 20 µm]. B- Leptocytes and codocytes are evident, characteristic findings of iron deficiency anemia [Diff-Quick stain ×1000; Scale bar = 5 µm].

Serum biochemistry [Catalyst One][5] showed mildlyincreasedactivities of alkaline phosphatase, gamma-glutamyl transferase, aspartate aminotransferase, and alanine aminotransferase. Coagulation profiles [Coag Dx][5] revealed prothrombin time and activated partial thromboplastin time within reference intervals. Fecal microscopic examination, whole blood quantitative PCR [Anemia Real PCR Panel-Canine][5], genetic testing for pyruvate kinase[7] saline agglutination test and Coombs' test were all negative. Thoracic radiographs revealed a soft tissue mass at the distal esophagus near the diaphragmatic hiatus, whereas abdominal radiographs, abdominal ultrasonography, and urinalysis were unremarkable.

Capsule endoscopy[8] identified multiple tortuous and dilated blood vessels at the gastroesophageal junction [GEJ], suggestive of AGD, which was subsequently confirmed by flexible endoscopy (Figure 2A & B). Computed tomography revealed a well-defined extraluminal mass [3.8 × 1.6 × 2.8 cm; length × width × height] adjacent to the GEJ, circumferentially involving the distal esophagus (Figure 3). No additional abnormalities, including ulceration, were identified in the esophagus, stomach, small intestine, or colon. Based on these findings, the patient was definitively diagnosed with AGD associated with an esophageal mass, which was the cause of the GI bleeding and iron-deficiency anemia.

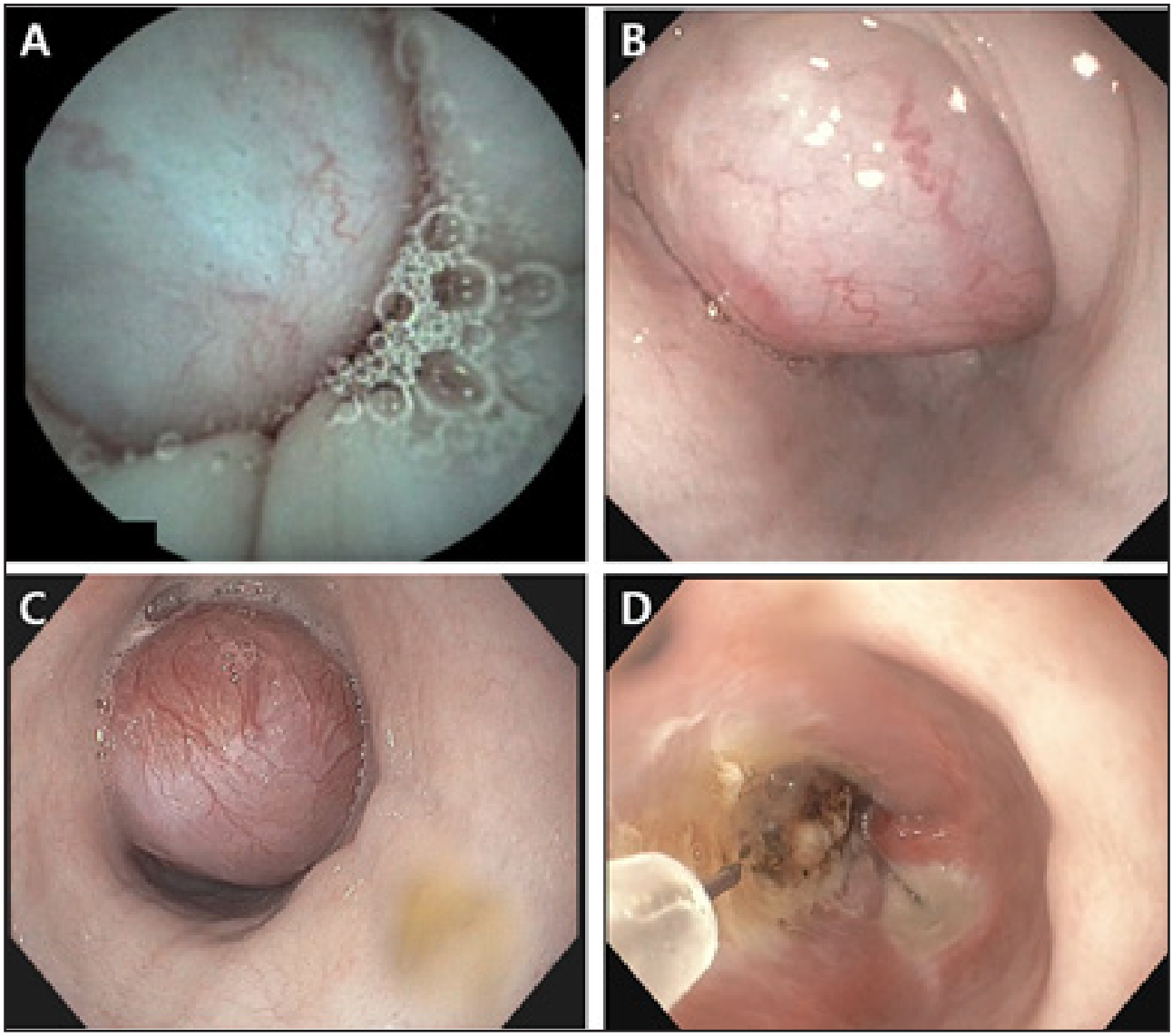

Figure 2. Endoscopic findings of gastroesophageal junction (GEJ). A- Capsule endoscopy reveals multiple tortuous and dilated mucosal vessels at the GEJ. B- Flexible endoscopy shows angiodysplasia (AGD) at the GEJ, externally compressed by an adjacent extraluminal esophageal mass. C- Recurrent intraluminal mass at the same site. D- After mass resection, no evidence of AGD is observed.

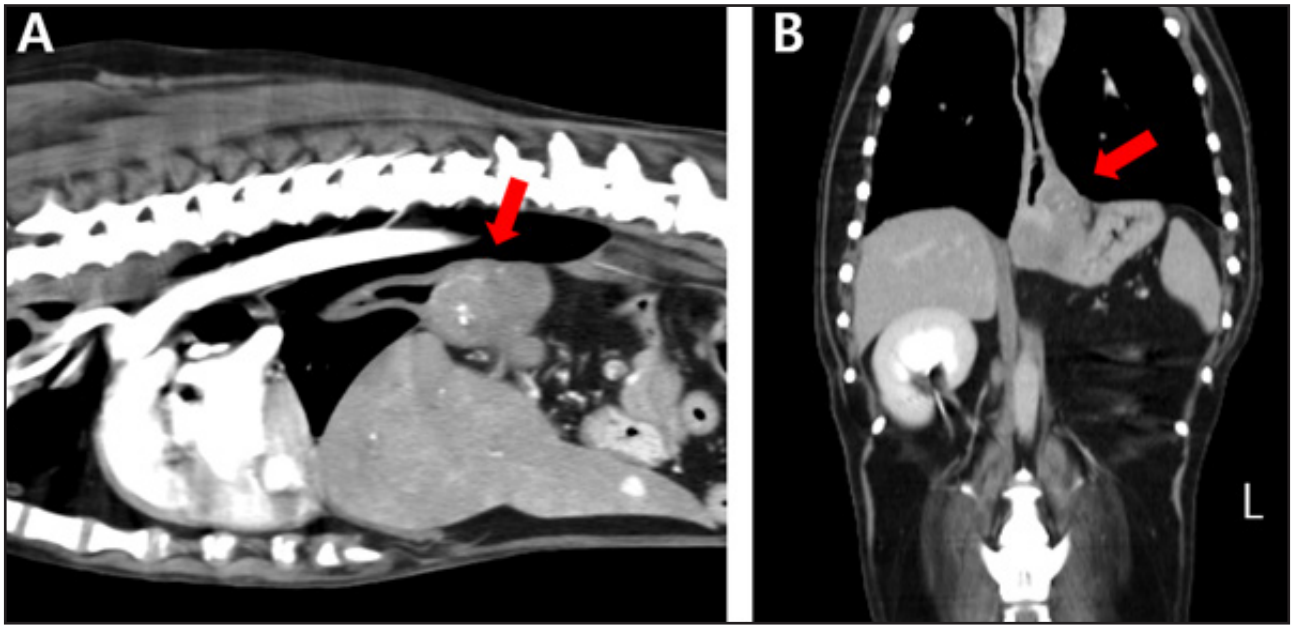

Figure 3. Computed tomography [CT] images. A- Sagittal & B- dorsal contrast-enhanced CT images showing an extraluminal mass [red arrow] adjacent to the GEJ, circumferentially involving the esophagus

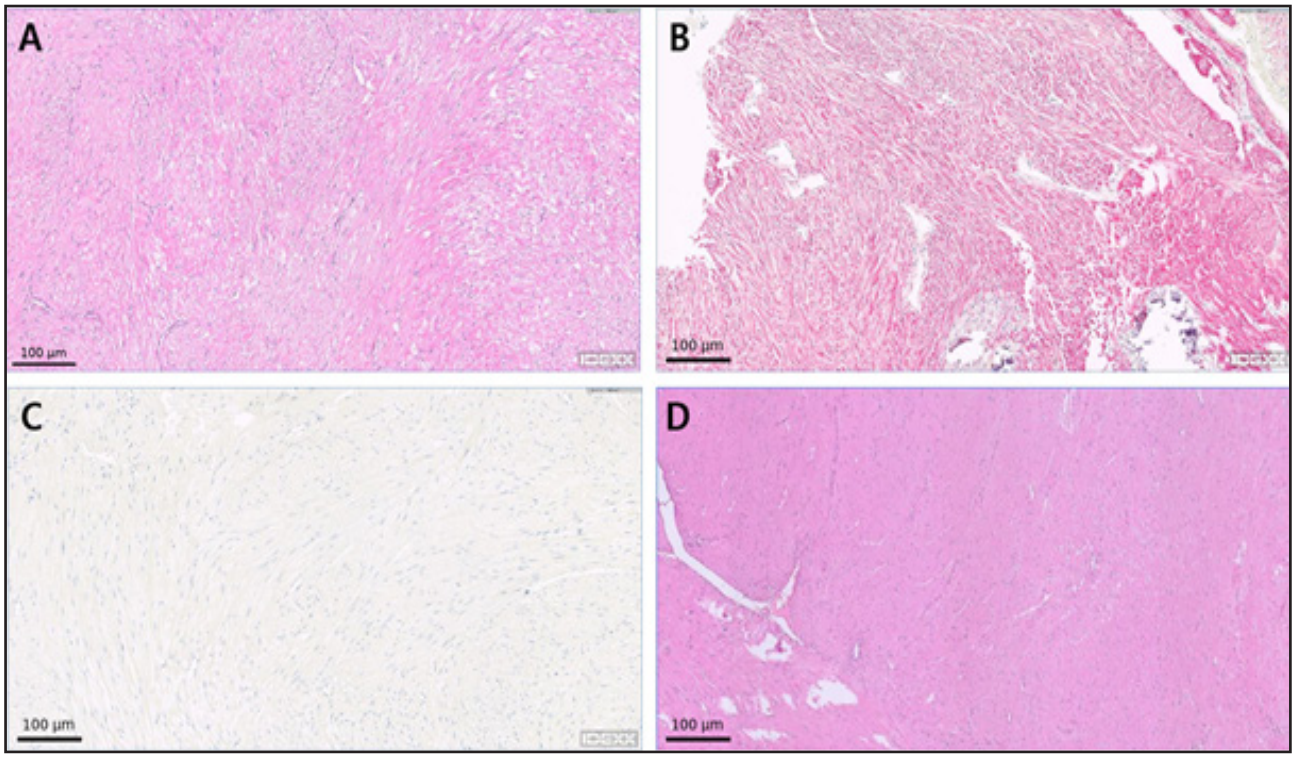

Figure 4. Histopathologic and immunohistochemical (IHC) findings of the esophageal mass. A- Histopathology of the initial incisional biopsy reveals a densely cellular spindle cell tumor with mild pleomorphism [H&E stain ×200; Scale bar = 100 µm]. B- IHC demonstrates strong cytoplasmic positivity for smooth muscle actin [×200; Scale bar = 100 µm]. C- The neoplastic cells are negative for c-Kit and DOG1, ruling out gastrointestinal stromal tumor and confirming smooth muscle origin, consistent with leiomyoma [IHC ×200; Scale bar = 100 µm]. D- Histopathology of the recurrent mass shows well-demarcated spindle cell proliferation with mild anisocytosis and anisokaryosis, again consistent with leiomyoma [H&E stain ×200; Scale bar = 100 µm].

Hormonal therapy with ethinyl estradiol [2.3 µg/kg - orally once daily] and levonorgestrel[9] [12 µg/kg - orally once daily] was initiated anda whole blood transfusion [16 mL/kg] was administered, leading to an increase in HCT to 28%. Ten days after the initiation of hormonal therapy, the HCT increased to 41.4%. On the same day, surgical debulking of the mass was performed to provide partial decompression of the esophagus. Histopathology and immunohistochemistry[5] demonstrated spindle cells positive for smooth muscle actin and negative for c-Kit and DOG1, confirming esophageal leiomyoma (Figure 4). Finally, the esophageal AGD was considered to be a consequence of extraluminal leiomyoma.

Hormonal therapy was maintained for a total of 93 weeks, during which the dog remained clinically stable without recurrence of anemia, AGD, or adverse effects related to hormonal therapy. At 53 weeks after the initiation of hormonal therapy, repeat endoscopy revealed recurrence of the intraluminal mass at the same site, without evidence of AGD (Figure 2C & D). The mass was endoscopically removed using an endoscopic snare and electrocautery[10], and histopathologic evaluation confirmed recurrent leiomyoma. The patient was subsequently lost to follow-upat approximately 95 weeks after initiation of hormonal therapy.

DISCUSSION

AGD is recognized as an uncommon vascular malformation characterized by abnormal dilatation and tortuous mucosal and submucosal vessels that can lead to chronic blood loss [15]. In humans, AGD is a relatively common cause of GI bleeding, accounting for approximately 4% of cases, with 77% of affected patients experiencing overt bleeding [15]. In contrast, reports of GI AGD in veterinary medicine are rare, with 23 canine cases described to date [3,13]. Clinical signs most often include intermittent hematochezia or melena and approximately 40% of affected dogs presented with iron-deficiency anemia [3].

The etiology of AGD remains unclear and is generally regarded as idiopathic in dogs. In humans,chronic, intermittent obstruction of submucosal veins is considered a key mechanism [15]. This is thought to occur particularly in regions of high wall tension such as the cecum and ascending colon, where repeated colonic distension increases intraluminal pressure and radius, intermittently blocking submucosal venous outflow according to the Law of Laplace [5,15]. Additional mechanical factors such as intermittent obstruction, transient rises in intraluminal pressure, or extrinsic compression may also impair venous outflow. Impaired venous outflow can result in vascular congestion, failure of precapillary sphincters, and the formation of fragile arteriovenous collaterals [15]. The esophageal bodyis generally a low-tension, compliant organ and is less susceptible to AGD [12]. In contrast, the proximal GEJ exhibits intrinsically high wall tension due to the overlapping anatomical structures of the lower esophageal sphincter and the crural diaphragm [10]. In the present case, an esophageal leiomyoma located adjacent to this anatomically high-tension site is hypothesized to have induced AGD through chronic extrinsic mechanical compression. This mechanism likely led to intermittent obstruction of submucosal venous drainage, fostering the development of the angiodysplastic lesions.

While definitive resection of esophageal leiomyomas can be curative, it is often not feasible due to the tumor's extent and critical location [1,17]. Specifically, resection of a caudal esophageal mass may require gastric advancement, which carries the risk of persistent gastroesophageal reflux and esophagitis [1]. Given this unfavorable risk-benefit profile inherent to surgery at the GEJ, we opted for surgical debulking rather than complete excision and focused on a conservative bleeding-control strategy for the associated AGD. Management of AGD in human medicine primarily targets the bleeding lesion. Endoscopic ablation techniques, such as argon plasma coagulation, clipping, or laser photocoagulation, are considered 1st-line treatments [15]. Surgical resection is reserved for uncontrolled acute hemorrhage refractory to other therapies, owing to the risk of morbidity and recurrence [15]. Pharmacologic agents, including thalidomide and octreotide, have shown efficacy in refractory or diffuse lesions through anti-angiogenic mechanisms [15]. Hormonal therapy with estrogen-progesterone combinations has also been used in humans; however, its efficacy remains controversial due to inconsistent outcomes[9]. The mechanism of hormonal therapy remains unclear; however, proposed theories include induction of intestinal epithelial metaplasia with development of protective keratinizing squamous epithelium, restoration of endothelial continuity, and modulation of coagulation pathways [18].

In veterinary medicine, only few AGD cases have been reported treated with surgical resection, endoscopic ablation, and hormonal therapy. However, invasive approaches carry significant risks such as septic peritonitis after subtotal colectomy or colonic perforation after endoscopic ablation [7,11]. Clinical outcomes with hormonal treatment have been variable. Among 4 canine cases, 2 achieved remissions lasting 8 and 24 months, 1 showed no response, and 1 improved within 6 weeks with anemia resolving by 12 months [6,7,13]. In the present case, anemia resolved within 10 days after initiation of hormonal therapy, which was continued for 93 weeks. The sustained remission of AGD despite tumor recurrence supports the efficacy of long-term hormonal therapy in reducing vascular fragility and bleeding risk and subsequent surgical debulking further improved clinical control. Hormonal protocols in previous canine reports varied, most commonly combining ethinyl estradiol with norethindrone acetate, or used diethylstilbestrol with altrenogest [6,7,13]. In our case, a combination of ethinyl estradiol [2.3 µg/kg orally once daily] with levonorgestrel [12 µg/kg - orally once daily] was administered due to the unavailability of norethindrone acetate in South Korea. The dose of levonorgestrel used was approximately 1/100 of the previously reported dose [1 mg/kg - orally once daily] that produced only mild, clinically insignificant androgenic effects [8]. In addition, ethinyl estradiol appears safe in dogs at doses below 40-200 µg/kg daily, the reported no-observed-effect level for hematotoxicity [6]. No adverse effects were observed during nearly 2 years of continuous administration, supporting safety of long-term hormonal therapy when used at appropriate doses.

To our knowledge, this is the 1st report of esophageal AGDassociated with an esophageal leiomyoma in a dog. Chronic venous outflow obstruction from extrinsic compression at the high-tension GEJ provides a plausible mechanism linking the tumor to AGD. When definitive surgical intervention is limited by high-risk locations like the GEJ, adjunctivelong-term hormonal therapy may be considered a strategic bleeding-control option for managing AGD. Long-term hormonal therapy, combined with surgical debulking, was safe and effective, providing remission for almost 2 years.

MANUFACTURERS

[1]Yuhan Co. Ltd. Seoul, Republic of South Korea.

[2]LaRoche Ltd. Basel, Switzerland.

[3]Sanofi S.A. Paris, France.

[4]Bayer A.G. Leverkusen, Germany.

[5]IDEXX Laboratories. Inc. Westbrook, ME, USA.

[6]Neodin BioVet Laboratory. Seoul, Republic of South Korea.

[7]Korea Vet Lab. Seoul, Republic of South Korea.

[8]Intromedic Co. Ltd. Seoul, Republic of South Korea

[9]Hana Pharm Co. Ltd. Seoul, Republic of South Korea.

[10]Olympus Medical Systems. Tokyo, Japan.

Acknowledgements. This work was supported by the National Research Foundation of Korea (NRF) grant funded by the Korea government (MSIT) (RS-2026-25483764) and the Basic Science Research Program through the NRF funded by the Ministry of Education (RS-2023-00219710314820926400 01).

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 **Amsellem P.M. & Farese J.P. 2019.** Esophageal Tumors. In: Vail D.M., Thamm D.H. & Liptak J.M. (Eds). *Withrow & MacEwen's Small Animal Clinical Oncology*. 6th edn. St. Louis: Elsevier, pp.449-450.

2 **Daugherty M.A., Leib M.S., Lanz O.I. & Duncan R.B. 2006.** Diagnosis and surgical management of vascular ectasia in a dog. *Journal of the American Veterinary Medical Association*. 229(6): 975-979.

3 **Defarges A., Stiller J. & Solomon J.A. 2023.** Gastrointestinal angiodysplasias diagnosed using video capsule endoscopy in 15 dogs. *Journal of Veterinary Internal Medicine*. 37(2): 428-436. DOI: 10.1111/jvim.16677

4 **Fan T., Simpson K., Polack E., Dykes N. & Harvey J. 1999.** Intestinal haemorrhage associated with colonic vascular ectasia [angiodysplasia] in a dog. *Journal of Small Animal Practice*. 40(1): 25-30. DOI: 10.1111/j.1748-5827.1999.tb03184.x

5 **Foutch P.G. 1993.** Angiodysplasia of the gastrointestinal tract. *American Journal of Gastroenterology [Springer Nature]*. 88(6): 807-818.

6 **Goldman C., Parnell N. & Holland C. 2008.** Hormone therapy for treatment of colonic vascular ectasia in 2 dogs. *Journal of Veterinary Internal Medicine*. 22(4): 1048-1051. DOI: 10.1111/j.1939-1676.2008.0102.x

7 **Harris A.N., Armentano R.A., Torres A.R. & Gallagher A.E. 2016.** Use of endoscopic-assisted argon plasma coagulation for the treatment of colonic vascular ectasia [angiodysplasia] in an adult dog. *Journal of the American Veterinary Medical Association*. 248(5): 526-531. DOI: 10.2460/javma.248.5.526

8 **Hillesheim H., Hoffmann H., Güttner J. & Oettel M. 1982.** Toxicity of the Progestagen STS 557 Compared toLevonorgestrel in Beagles After Oral Administration for 6 Months. In: Chambers P.L. & Chambers C.M. (Eds). *New Toxicology for Old: A Critique of Accepted Requirements and Methodology*. Berlin: Springer-Verlag, pp.221-224..

9 **Junquera F., Feu F., Papo M., Videla S., Armengol J.R., Bordas J.M., Saperas E., Piqué J.M. & Malagelada J.R. 2001.** A multicenter, randomized, clinical trial of hormonal therapy in the prevention of rebleeding from gastrointestinal angiodysplasia. *Gastroenterology*. 121(5): 1073-1079. DOI: 10.1053/gast.2001.28665

10 **McMahon B., Frøkjær J.B., Drewes A.M. & Gregersen H. 2004.** A new measurement of oesophago-gastric junction competence. *Neurogastroenterology and Motility*. 16(5): 543-546. DOI: 10.1111/j.1365-2982.2004.00543.x

11 **Nemeth T., Solymosi N. & Balka G. 2008.** Long-term results of subtotal colectomy for acquired hypertrophic megacolon in eight dogs. *Journal of Small Animal Practice*. 49(12): 618-624. DOI: 10.1111/j.1748-5827.2008.00609.x

12 **Orvar K.B., Gregersen H. & Christensen J. 1993.** Biomechanical characteristics of the human esophagus. *Digestive Diseases and Sciences*. 38(2): 197-205. DOI: 10.1007/BF01307545

13 **Paulin M.V., Cosford K., Sato-Takada K., Koo S.T. & Carr A. 2022.** Successful medical management of an acute life-threatening haemorrhagic crisis related to acquired colonic vascular ectasia in a dog. *Veterinary Record Case Reports*. 10(4): e497. DOI: 10.1002/vrc2.497

14 **Rogers K.S., Butler L.M., Edwards J.F., Brassard J., Boothe H. & Cargile J. 1992.** Rectal hemorrhage associated with vascular ectasia in a young dog. *Journal of the American Veterinary Medical Association*. 200(9): 1349-1351.

15 **Sami S.S., Al-Araji S. & Ragunath K. 2014.** gastrointestinal angiodysplasia–pathogenesis, diagnosis and management. *Alimentary Pharmacology and Therapeutics*. 39(1): 15-34. DOI: 10.1111/apt.12529

16 **Tomita T., Palacherla J., Zuckerman M., Dougherty S. & Ghaleb M. 2004.** Case report: gastrointestinal stromal tumor of jejunum with angiodysplasia. *Digestive Diseases and Sciences*. 49(4): 667. DOI: 10.1023/B:DDAS.0000026328.92332.e6

17 **Turner A.I. 2017.** Cancer of the esophagus and stomach. In: Ettinger S.J., Feldman E.C., Côté E. (Eds). *Textbook of Veterinary Internal Medicine*. 8th edn. St. Louis: Elsevier, pp.1283-1286.

18 **Van Cutsem E., Rutgeerts P. & Vantrappen G. 1990.** Treatment of bleeding gastrointestinal vascular malformations with oestrogen-progesterone. *The Lancet*. 335(8695): 953-955. DOI: 10.1016/0140-6736(90)91022-D