Life-threatening Anemia in a Dog Caused by the Nile Leech, *Limnatis nilotica* (Hirudinidae) in Israel

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ABSTRACT

This is the second report of a naturally occurring internal hirudiniasis due to the Nile leech (*Limnatis nilotica*) in a dog in Israel. A 3.5-year-old Shih Tzu dog presented with lethargy, collapse, and severe, life-threatening hemorrhagic anemia. Upon initial physical examinations, at both the referring clinic and the hospital, a sublingual hematoma was erroneously diagnosed a sublingual hematoma. Nevertheless, comprehensive oral examination under general anesthesia revealed no evidence of hematoma, while only two engorged leeches attached to the sublingual mucosa, which were removed. Hemostatic tests showed several and significant abnormalities, previously unreported in similar cases, including prolonged clotting times, hyperfibrinolysis, indicated by increased D-dimer concentration, and decreased antithrombin activity, likely reflecting the systemic impact of the anticoagulants and other bioactive molecules found in leech saliva, which can disrupt the host's coagulation pathways beyond localized bleeding. The dog was administered a blood transfusion and supportive care, leading to full clinical recovery within three days. The final diagnosis was severe acute hemorrhagic anemia and hypoproteinemia caused by the leech infestation. This report underscores the critical importance of including leech infestation among the differential diagnoses for unexplained acute anemia in dogs, particularly in endemic areas with potential exposure to leech-contaminated water sources. It highlights that leeches might be easily misidentified as hematomas during routine physical examinations, warranting thorough oral and pharyngeal examinations, ideally under sedation or anesthesia, to ensure accurate diagnosis and timely intervention. The unique hemostatic abnormalities observed herein also warrant further investigation into the systemic hemostatic effects of leech saliva.

Keywords: Hirudiniasis; Canine; Hemostasis; Coagulation; Hyperfibrinolyisis; Antithrombin.

INTRODUCTION

Leeches (phylum: Annelida; subclass: Hirudinea) are parasitic, segmented worms, comprising 680 species, distributed worldwide (excluding in the Antarctica). Many are blood-sucking parasites, infesting vertebrates and invertebrates, while others are considered predators, and rarely, scavengers. Most leech species live in freshwater habitats, although some are marine and terrestrial species (1).

Leech infestation, termed hirudiniasis, usually occurs following host contact with leech-infested water, while drinking or swimming (2). Hirudiniasis is well described in both humans and animals. Internal hirudiniasis is a leech

infestation of internal organ or anatomic site. In humans, internal hirudiniasis most commonly involves the oral cavity. Its clinical signs vary, depending on the affected organ, which might include the nasal cavities, nasopharynx, larynx, vagina, and urinary bladder (3). Surprisingly, internal hirudiniasis was previously reported in only seven dogs, all involving the oral or nasal cavities (2, 4-8).

This report describes a case of oral hirudiniasis due to infestation with the Nile leech, *Limnatis nilotica*, leading to severe, life-threatening blood-loss anemia in an adult Shi-Tzu dog. To the best of the authors' knowledge, this is the second report of naturally-occurring internal hirudiniasis in domestic dogs in Israel (8). This report also includes novel hemostatic findings, previously unreported in similar cases in both humans and dogs.

CASE REPORT

A 3.5-year-old, 4.6-kg female Shih Tzu dog was presented to the Hebrew University Veterinary Teaching Hospital (HUVTH) Emergency Department (HUVTH-ED) due to oral bleeding. Previous medical history included bilateral corneal perforation seconry to trichiasis and self-mutilation, which was surgically treated at the HUVTH. Three days before the current presentation, the dog was presented for a scheduled recheck to the HUVTH Ophthalmology Department (OD), was bright and alert, and showed severe pruritus and flea infestation. On that recheck, the owner had mentioned that two days previously, a dry cough was noted, and therefore, the dog was examined by the primary care veterinarian. It was tentatively diagnosed with kennel cough. No treatment was prescribed. Since then, the cough had almost completely subsided. The dog was discharged from the HUVTH-OD. For the ophthalmic problem, the flea infestation and the pruritus, ophthalmic topical neomycin-polymyxin B-dexamethasone ointment (Maxitrol, Novartis Pharma AG, Basel, Switzerland; q12h), oclacitinib (Apoquel, Zoetis, Kalamazoo, MI; 2.7 mg q12h for 7 days PO), and fluralaner (Bravecto, Intervet, Vienna, Austria; 250 mg PO, once), were prescribed, and an Elizabethan collar was applied.

Post-discharge, the cough persisted, occurring once daily, while oral bleeding was noted at the end of a coughing episode. Three days after the previous presentation to the HUVTH, the dog became lethargic and depressed, collapsed during walks, and blood stains were noted on its pillow. It was then represented to the primary care clinic. Physical examination revealed oral bleeding, and a sublingual hematoma was suspected. CBC performed by the referring clinic (Table 1) demonstrated severe anemia (hematocrit, 14.7%; reference interval [RI], 36.9-60), neutrophilic leukocytosis, and platelet count within RI. Total plasma protein (TPP) concentration, measured by refractometry was 3.8 g/dL (RI, 5.5-7.5). Serum chemistry abnormalities included marked hypoalbuminemia, hypoglubulinemia, marked hypoproteinemia, and mild total hypocalcemia (Table 1). Microscopic blood smear examination at the referring clinic showed neutrophilic leukocytosis with mature non-toxic neutrophils, marked anisocytosis, polychromasia, and metarubricytosis, indicative of regenerative anemia. The metarubricytosis probably led to an erroneously high lymphocyte count by the hematology analyzer. It was recommended to leave the sublingual hematoma untreated, to be spontaneously resorbed. Blood transfusion was recommended, but was declined by the dog owner. Hence, the dog was discharged. At home, the dog showed lethargy, tachypnea, dyspnea, and bilateral mucous nasal discharge. It was therefore presented to the HUVTH-ED.

At presentation, the dog was quiet, alert and responsive, with tachycardia (heart rate, 180 bpm), tachypnea (respiratory rate, 36 breaths/min), a normal rectal temperature, pale mucous membranes, melena upon rectal examination, and a dark red sublingual hematoma. Point-of-care abdominal sonography was unremarkable.

CBC (Advia 2120i, Siemens, Erfurt, Germany) showed neutrophilic leukocytosis (white blood cells [WBC], $28x10^3/\mu L$; RI, 5.2-13.9x10^3/microL) severe macrocytic, hypochromic, markedly regenerative (reticulocytes, 370.3 $x10^9/L$; RI, 8.4-129.3), anemia (hematocrit, 12.9%; RI, 37.1-57). The platelet count was within RI (Table 1). Blood smear (stained with modified Wright's staining solution) microscopic examination revealed metarubricytosis (13 nucleated red blood cells per 100 leukocytes). The WBC count, corrected for the presence of nucleated red blood cells was $24.64x10^3/\mu L$ (Table 1). The relative lymphocyte count was 3.45%. The neutrophils were mildly left-shifted, and neutrophilic bands occasionally showed mild cytoplasmic toxicity (basophilia). The RBC showed marked polychromasia and anisocytosis, moderate macrocytosis and mild hypochromia, with rare schistocytes and keratocytes. The metarubricytosis was deemed appropriate (physiologic). The

Table 1: Laboratory results at presentation to the referring clinic and to the hospital, and during hospitalization of a dog with severe life-threatening anemia caused by sublingual *Limnatis nilotica* leech infestation.

Analyte (units)	Referring clinic results (RI)	Results at presentation to the hospital (RI)	Day 1	Day 2
Leukocytes (x10^3/μL)	25.95 (4.0-16.5)	28.0[1] (5.2-13.9)		
Neutrophils (x10^3/μL)	16.43 (2.9-11.7)	23.86 (3.9-8.0)		
Monocytes (x10^3/μL)	0.15 (0.14-1.02)	0.71 (0.2-1.1)		
Lymphocytes (x10^3/μL)	9.2 (0.83-4.91)	3.14[2] (1.3-4.1)		
Eosinophils (x10^3/μL)	0.13 (0.04-1.62)	0.11 (0.0-0.6)		
Basophils (x10^3/μL)	0.04 (0-0.120	0.03 (0.0-0.1)		
Large unclassified cells (x10^3/μL)	ND	0.07 (0-0.3)		
nRBC	ND	3.22[3] (0.0-0.0)		
Red blood cells (x10^6/μL)	2.19 (5.8-8.9)	1.6 (5.7-8.8)		
RDW (%)	23.7 (12.2-16.4)	23.1 (11.9-14.5)		
Hematocrit (%)	14.68 (36.9-60)	12.9 (37.1-57)		
Hemoglobin (g/dL)	4.7 (13.2-22.0)	3.4 (12.9-18.4)		
Mean corpuscular volume (fL)	67 (60-76)	80.1 (58.8-71.2)		
MCHC (g/dL)	31.8 (33.4-39.2)	26.7 (31-36.2)		
Reticulocytes (%)	ND	22.6 (0.1-2.0)		
Reticulocytes (x10^3/μL)	ND	370.3 (8.4-129.3)		
Platelets (x10^3/μL)	209 (126-660)	293 (143-400)		
Mean platelet volume (fL)	11.2 (8.0-14.1)	14.5 (7-11)		
Plateletcrit (PCT) (%)	0.23 (0.14-0.55)	0.43 (0.1-0.4)		
In saline slide agglutination		Negative		
RBC osmotic fragility		Negative		
Packed cell volume[4] (%)	14.7 (36.9-60)	13 (37-57)	22	25
Total plasma protein[5] (g/dL)	3.8 (5.5-7.5)	4.0 (5.5-7.5)	4.6	4.8
Serum albumin (g/dL)	2.4 (2.5-3.7)	(3.0-4.4)	2.8	
Serum creatinine (mg/dL)	0.4 (0.6-1.2)	(0.3-1.3)	0.22	
Serum globulins (g/dL)	1.7 (2.5-3.6)	(1.8-3.9)	1.8	
Serum total protein (g/dL)	4.1 (5.0-7.6)			
Serum total calcium (mg/dL)	8.8 (9.0-12.0)			
Prothrombin time (sec)		7.8 (6.0-8.4)	11.1	
aPTT (sec)		14.8 (11.0-17.4)	24.1	
Fibrinogen (mg/dL)		326 (200-400)	326	
Antithrombin activity (%)		(87-140)	55	
D-dimer (ng/mL)		(<250)	1509	

RI, reference interval; nRBC, nucleated red blood cells;
RDW, red blood cell distribution width;
MCHC, mean corpuscular hemoglobin concentration;
aPTT, activated partial thromboplastin time;
ND, not determined.
[1]The corrected leukocyte count for presence of nucleated red blood cells was 24.64x10^3/μL; [2]The corrected lymphocyte count for presence of nucleated red blood cells was 0.85x10^3/μL; [3]The nRBC count was calculated based on a manual differential count of 13 nRBC per 100 leukocytes in the blood smear; [4]Measured manually by centrifugation; [5]Measured in harvested plasma by refractometry.

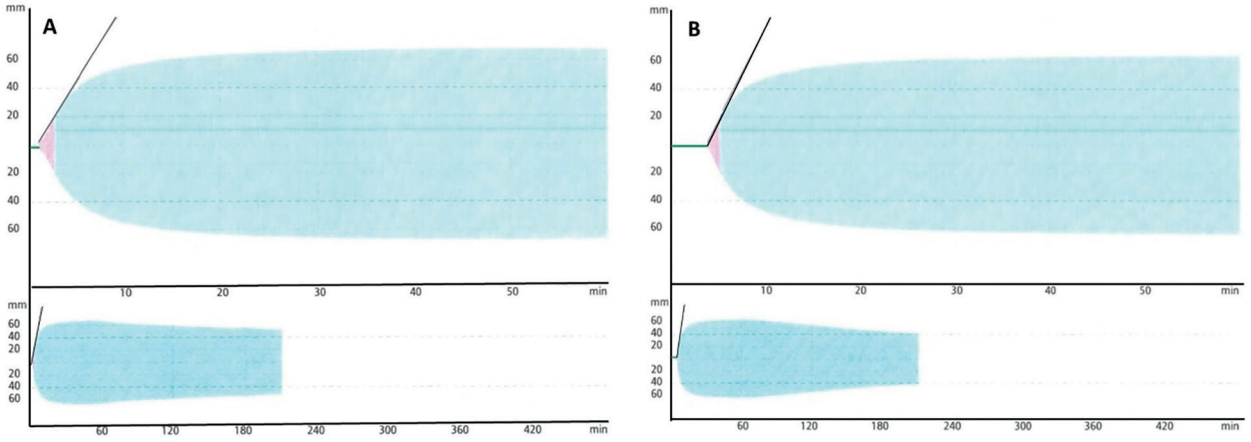

Figure 1: Extrinsic (A) and intrinsic (B) thromboelastometry (ExTEM and InTEM, respectively) tracings on Day-2 of hospitalization of a dog infested sublingually by the Nile leech *Limnatis nilotica,* showing increased lysis.

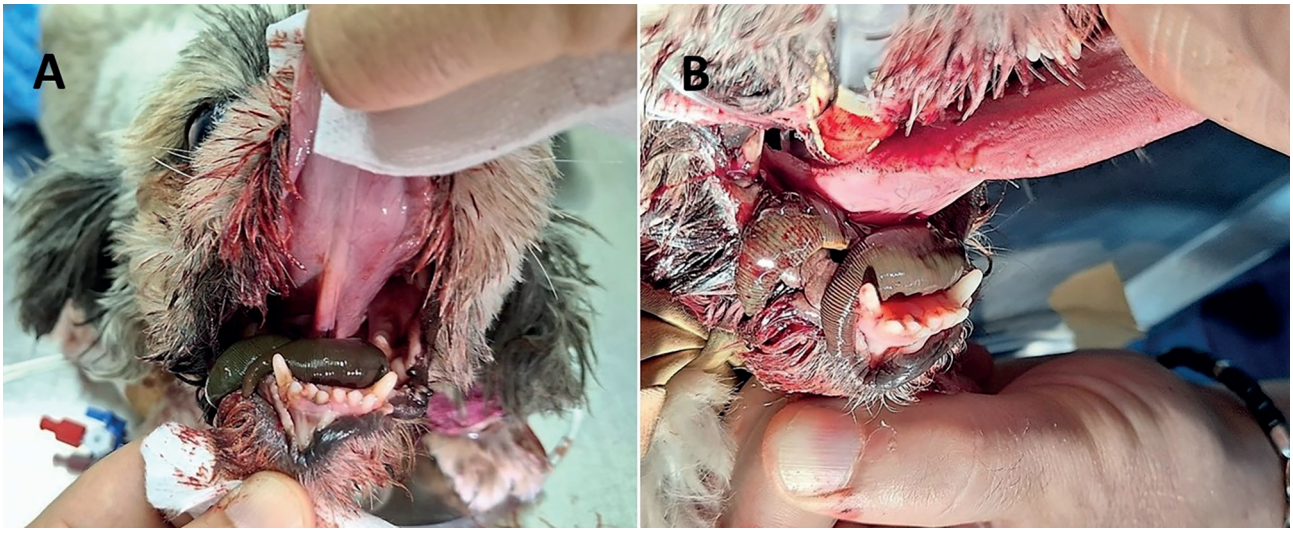

Figure 2: Two *Limnatis nilotica* leeches attached to the sublingual mucosa, engorged with blood and protruding to right side of the mouth in a dog, upon examination under heavy sedation. Note the bloody discharge on the hair coat and the ventral aspect of the tongue.

automated platelet count was confirmed. TPP (measured by refractometry; Atago, Tokyo, Japan) was 4.0 g/dL (RI, 5.5-8.5) (Table 1). The dog's blood type was DEA-1.1-negative. The prothrombin time (PT) and activated partial thromboplastin time (aPTT) (ACL Top 300, IL, Milano, Italy) were within the RIs. In-saline slide agglutination and RBC osmotic fragility tests were negative.

Initial therapy included a DEA-1.1-negative packed RBC transfusion (100 mL IV over 4 hours, at 21 mL/kg), which led to rapid clinical improvement and voluntary eating. Soon after the meal, severe oral bleeding was noted. Thus, the oral cavity was examined under mild sedation with butorphanol (Butomidor, Vetviva, Wels, Austria; 0.2 mg/kg, IV) and midazolam (generic, AS Klceks, Krustpils Iela, Riga, Latvia; 0.2 mg/kg, IV). A sublingual mass was noted, which was again assessed as a hematoma, while the rest of the oral cavity appeared unremarkable. The owners declined thoracic radiographs and further laboratory testing due to financial constraints. The clinical assessment then was that the anemia was acute hemorrhagic, due to bleeding from the oral hematoma, and possibly also from gastrointestinal bleeding, both resulting in melena. The post-blood transfu-

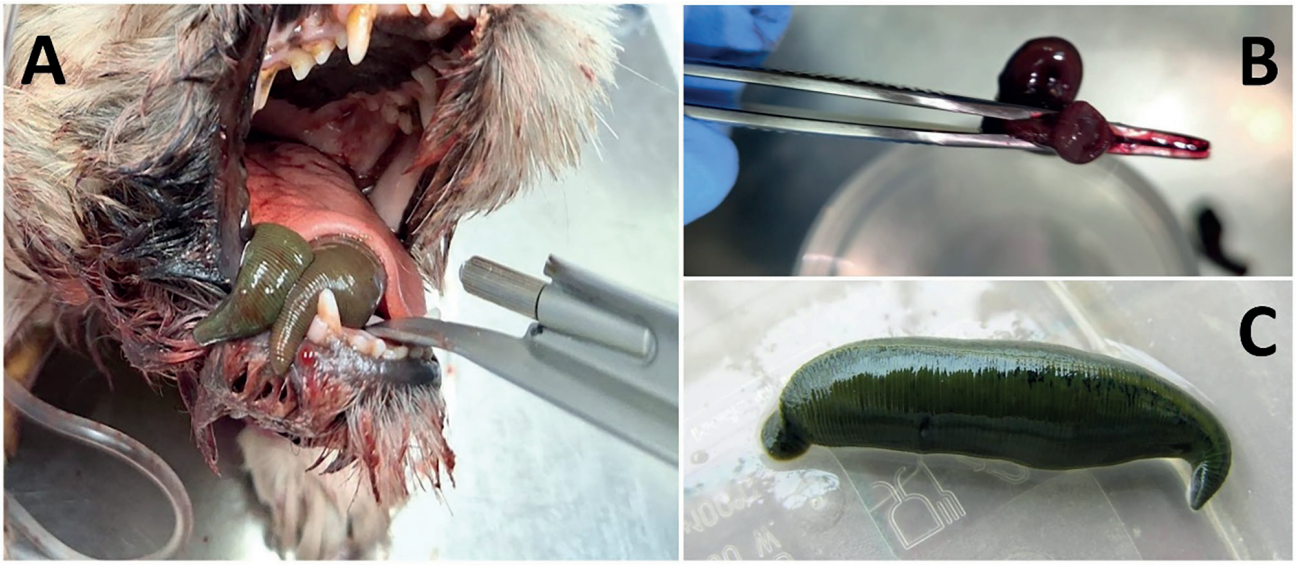

Figure 3: A. Two *Limnatis nilotica* leeches attached to the sublingual mucosa immediately before detaching them. Salt was applied on the leaches, and they were gently pulled and detached using forceps (**B**), and were still viable (**C**).

sion packed cell volume (PCV) and TPP were 28% and 5.0 g/dL, respectively (Table 1).

During hospitalization, the supportive treatment included IV lactated Ringer's solution (Bieffe Medical SA, Sabinarigo, Spain), with metoclopramide (Pramin, RAFA laboratories, Jerusalem, Israel; 1 mg/kg/day IV at constant rate infusion [CRI]), maropitant (Cerenia, Zoetis, Kalamazoo, MI; 1 mg/kg IV q24h), metronidazole (generic, B Braun, Melsungen, Germany; 25 mg/kg IV, g12h) and doxycycline (Doxylin, Dexcel, Or-Akiva, Israel; 5 mg/kg PO once). With the suspected gastrointestinal bleeding, possibly due to ulceration, famotidine (generic, Mylan, Rockford, IL; 8 mg/kg/day CRI IV), pantoprazole (generic, Hainan poly pharm Co., China; 1 mg/kg IV q12h) and sucralfate (VetMarket, Shoham, Israel; 500 mg PO, once) were also administered. PCV, TPP, blood glucose, arterial blood pressure, and vital signs were monitored.

On hospitalization day-2, the PCV was 22% and TPP was 4.6 g/dL (Table 1). Intrinsic and extrinsic thromboelastometry (INTEM and EXTEM, respectively; Rotem delta, Munich, Germany) demonstrated increased maximal lysis (ML; 34% and 22% respectively; RI, 0-15% for both) (Figure 1). Hemostatic test results included mildly prolonged PT and aPTT, normo-fibrinogenemia, increased D-dimer concentration, and decreased antithrombin activity (ATA) (Table 1).

Under general IV anesthesia, with butorphanol (0.2 mg/kg), midazolam (0.2 mg/kg) and propofol (generic, Fresenius Kabi, Bad Homburg, Germany; 5 mg/kg), comprehensive oral cavity examination and upper gastrointestinal endoscopy were done. While examining the oral cavity, before tracheal intubation, two engorged leeches were observed attached to the sublingual mucosa (Figure 2), while no actual hematoma was noted. Salt was applied to the leeches, and within minutes, they were gently pulled from the sublingual mucosa using forceps (Figure 3), which led to some mucosal bleeding. The assessment then was that these blood-engorged leeches, attached to the sublingual tissue, had actually been previously erroneously considered a large sublingual hematoma, both upon examinations in the primary care clinic and in the initial examinations at the HUVTH-ED. The dog then underwent tracheal intubation and upper gastrointestinal endoscopy, showing only some free blood remnants from the oral bleeding after pulling the leeches. The esophageal, gastric, and duodenal mucosae were unremarkable, ruling out upper gastrointestinal ulcerations. Recovery from general anesthesia was uneventful.

The leeches were identified as *Limnatis nilotica* based on the characteristic appearance previously described by Bromley (9). After identifying the leeches, the owners were specifically questioned as to travels to fresh water sources possibly infested with leeches. Apparently, 10 days before presentation to the HUVTH, the owners traveled with the dog to a spring in the Samaria area (Ayn Almasaraj [Ein

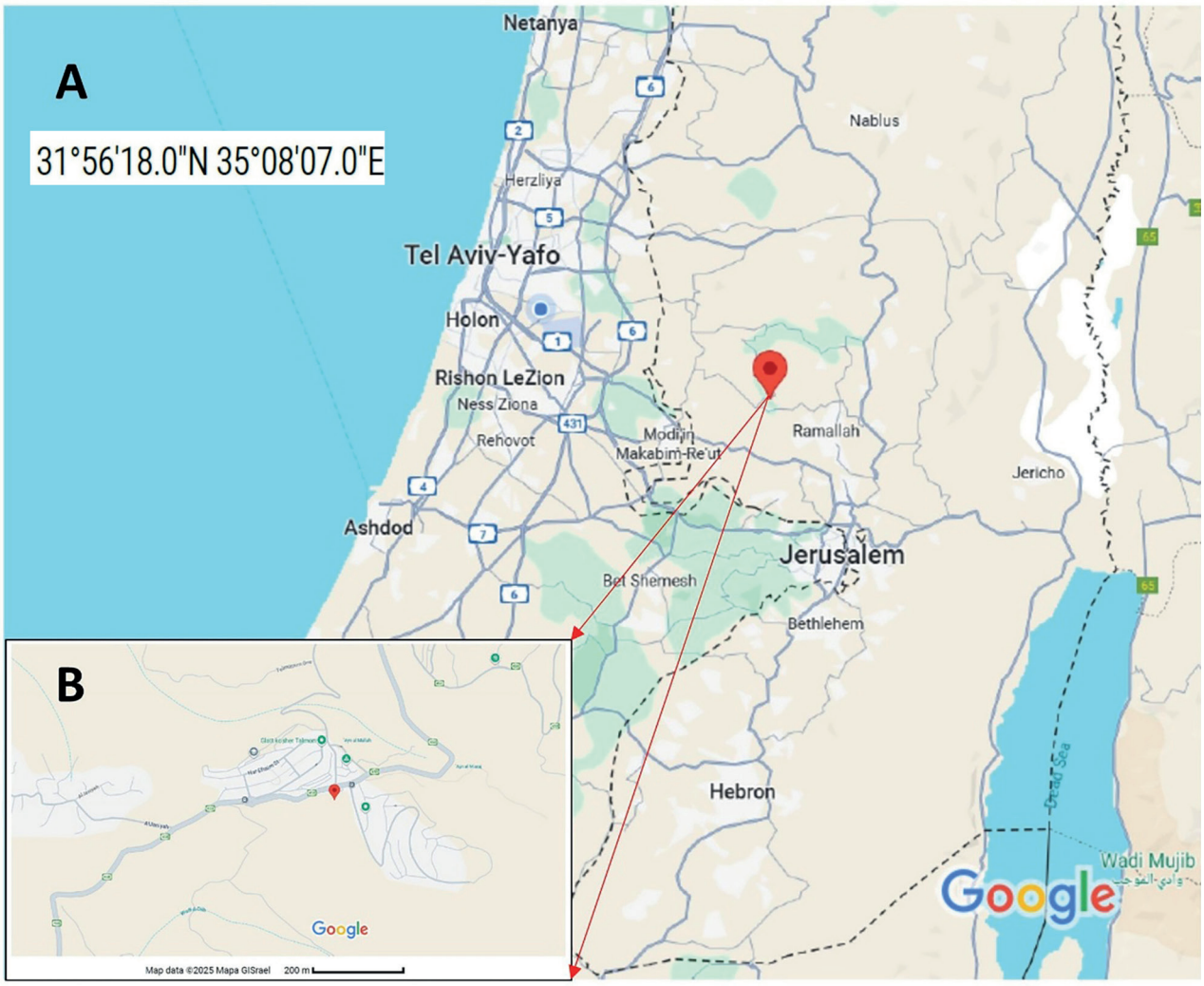

Figure 4: The coordinates and the geographical location of Ayn Almasaraj [Ein Talmon spring], where the dog was infested with Nile leeches (*Limnatis nilotica*). **B.** A higher magnification of the geographical location of the spring. The maps were generated using Google Maps and Map data 2025 Mapa, GISrael, Google.

Talmon]); 31°56′18″N 35°08′07″E; Figure 4). The coughing and oral bleeding were first noted several days after this trip. Around the same dates, an 8-year-old child was also infected with a leech attached to his soft palate at that same spring (10).

The final diagnosis and assessment were that the sublingual *L. nilotica* hirudiniasis resulted in severe acute hemorrhagic anemia, hypoproteinemia, shock and collapse, while coughing was deemed secondary to the oral bleeding.

The dog was discharged 24 hours post-presentation. Upon recheck, three days post-discharge, it was bright, alert, and responsive, with normal vital signs and no cough or oral bleeding. The PCV (23%) and TPP (5.8 g/dL) had improved. The PT and aPTT were mildly prolonged (11.1 sec and 24.1 sec, respectively). Although additional rechecks for monitoring of the anemia and the hemostasis were recommended, the dog was never represented and was thus lost to follow-up.

DISCUSSION

To the best of our knowledge, this is the second report of canine hirudiniasis in Israel. The first report was a brief parasitological note of oral hirudiniasis in a dog, with a single *L. nilotica* leech noted attached to the base of the tongue. The geographic location was not mentioned, and clinically, the only information reported was mucosal pallor (8). *L. nilotica*,

a freshwater leech, is typically attracted to animals and humans when they contact fresh water (e.g., ponds, streams, or pools and waterholes), entering the body orally or nasally, then attaching mostly to the pharyngeal, laryngeal, or nasal passages mucous membranes, and rarely they might invade the eyes, urethra, or vagina (11).

Leech infestation of dogs, or canine hirudiniasis, is rare but clinically a significant parasitism, typically resulting from ingestion or contact with aquatic leech-contaminated fresh water. Similar to the present dog, two mixed-breed puppies, reported from Iran, were presented with anorexia, hypersalivation, retching, oral bleeding, and microcytic, hypochromic anemia; live *L. nilotica* leeches were removed from the ventral tongue, leading to hematological normalization one month later (6). *L. nilotica* infestation was documented following a fall into a canal in Italy of a stray dog, exhibiting oral bleeding and exhaustion; The leech was removed by rhinoscopy, which led to recovery (5). Chronic nasal hirudiniasis with recurrent epistaxis in a dog was also successfully managed through nasal leech removal via rhinoscopy (4). In all the above previously reported cases in dogs, infestation likely occurred via ingesting unfiltered water. Clinical signs often included oral or nasal bleeding, pallor due to anemia, retching, hypersalivation, and sometimes respiratory distress (2-8). The diagnosis typically relies on direct leech visualization during examination, or endoscopy, followed by mechanical removal using forceps, aided by topical agents such as vinegar, saline, hypertonic saline, or salt (6). Secondary effects, including anemia and ongoing hemorrhage, due to hirudin and other anticoagulants in leech saliva, resolve with parasite removal and supportive care.

L. nilotica has a wide geographical distribution across Southern Europe (e.g., Mediterranean countries, Portugal, as far west as the Azores, Bulgaria, and Romania, the Middle East, Iran, Tajikistan, Central Asia, and North Africa). It is considered the most widely distributed leech species in Egypt, Israel, and Lebanon (11). In Israel, *L. nilotica* is widely but sporadically distributed, reported in both natural and man-made water sources, from the Negev Desert, where it is the only leech species documented, through the Judean Hills, and extending northward to the Galilee, Golan Heights, and the coastal plain (9).

Morphologically, *L. nilotica* is characterized by an elongated, dorsoventrally flattened body, typically dark green to brown with faint orange stripes. Smaller specimens tend to be paler in color than larger ones. Adults usually measure 30-100 mm in length (9). The jaws of this species are small, rounded, and relatively soft, equipped with approximately 30-40 coarse-surfaced teeth. While *L. nilotica* is incapable of penetrating intact skin, it can lacerate mucosal surfaces (e.g., oral, nasal, and pharyngeal), and occasionally invades the eye, urethra, and vagina (12). Mammals constitute the primary hosts, although incidental attachment to amphibians, such as frogs, has been reported.

Following attachment, *L. nilotica* uses its anterior sucker to adhere to soft tissues of the oral or buccopharyngeal cavities, while its body remains mobile (13,14). In heavy infestations, particularly among animals, multiple leeches might localize in the gums, sublingual area, or upper respiratory tract (15-21). In humans, single leeches are most often found in the pharynx or upper airway, persisting for several days to weeks (22). During infestation, *L. nilotica* causes mechanical trauma and might extract blood volumes up to 890% its body weight, inducing anemia and local tissue damage (19). Swelling of the affected mucosa might result in dysphagia or respiratory distress. Sometimes, leeches might remain in the host's throat without inducing clinical signs. When swallowed into the stomach, leeches are rapidly destroyed by gastric acid (23). Clinical manifestations of *L. nilotica* hirudiniasis in humans might include laryngopharyngitis and a foreign-body sensation in the throat, described as halazoun syndrome, or marrara in the Middle East (24).

In the dog described here, the anemia was severe and life-threatening, and accompanied by coughing and hemoptysis. The dog was hemodynamically unstable, with tachycardia, and was assessed in shock. Additional laboratory abnormalities included neutrophilic leukocytosis and hypoproteinemia. In this dog, the hypoproteinemia is attributed to the severe blood loss, while the leukocytosis is attributed to concurrent stress, inflammatory response, and erythroid regeneration with metarubricytosis. Metarubricytosis increased the leukocyte count, as nucleated red blood cells are counted as leukocytes by automated hematology analyzers, as occurred in this case (25).

Hemostatic test abnormalities included prolonged clotting times, increased D-dimer concentration, and decreased ATA. In most cases of human hirudiniasis, hemostatic tests were not reported, and when measured, the PT and aPTT were within their RIs (26). The latter two hemostatic ab-

normalities in this dog, to the best of our knowledge, were never reported in dogs or humans with hirudiniasis or in experimental models of hirudiniasis, and are particularly interesting. Increased D-dimer concentration indicates excessive fibrinolysis, which is also supported by the thromboelastometry results herein. This, to the best of our knowledge, has never been reported in canine or human hirudiniasis. In both the InTEM and ExTEM tracings, the Maximal Lysis (ML) analyte, which measures the percent decrease in clot strength compared to the maximal clot firmness, thereby reflecting the extent of fibrinolysis, was below RI. This indicates that clot breakdown was excessive due to hyperfibrinolysis, as might be expected, given the anticoagulant properties of the leech saliva (27).

Leeches have evolved mechanisms to disrupt their hosts' coagulation, suppress nociception, and reduce inflammation, thereby preserving blood in a fluid state during its ingestion and prolonged digestion (28). These adaptations include the production of anticoagulants and anti-inflammatory compounds. Some coagulation inhibitors have been identified in leeches (28). Several mechanisms potentially led to the increase in D-dimer concentration in the present dog. First, the attachment of *L. nilotica* to the oral mucosa and its prolonged blood feeding, possibly caused some mucosal microtrauma, potentially leading to local or systemic activation of the coagulation cascade, which later possibly resulted in hemostatic derangement and low-grade disseminated intravascular coagulation (DIC). This might be supported by the concurrently observed prolonged clotting time, increased D-dimer concentration (29), and decreased ATA. Nevertheless, occurrence of DIC in this dog cannot be completely supported in light of the normo-fibrinogenemia and the other thromboelastometry results, demonstrating normocoagulability. Unfortunately, a platelet count at this time point was unavailable, although the platelet count was within RI at presentation. The PT and aPTT prolongation might have merely reflected the direct antithrombin action of a hirudin-like substance in the leech saliva, which was possibly more pronounced in such a small dog.

Secondly, we propose that an inflammatory response to the leech infestation possibly also contributed to the hemostatic derangement by triggering pro-inflammatory cytokine (e.g., IL-6 and TNF-α) release, and upregulating endothelial cell and monocyte tissue factor expression, thereby promoting coagulation and fibrinolysis. The formation and subsequent breakdown of microthrombi resulted in increased D-dimer concentration (30). Thirdly, leech saliva contains several bioactive molecules, including anticoagulants (e.g., hirudin-like substances, acting as direct antithrombins), fibrinolytics (e.g., hementin, which cleaves fibrinogen), and vasodilators (e.g., histamine-like compounds), which might disrupt the local hemostatic balance, enhancing fibrin degradation and contributing to increased D-dimer concentration (27). Future studies of hirudiniasis cases, where comprehensive hemostatic tests are to be made, might establish whether increased D-dimer concentration is a consistent component of the hemostatic alterations induced by leech infestation.

The decreased ATA in the present dog, might have resulted from several mechanisms, including: 1) Low-grade DIC (as mentioned above), with antithrombin consumption, as it neutralizes excess thrombin (31); 2) Inflammation, leading to decreased hepatic antithrombin synthesis and its increased loss to the extravascular space because of increased vascular permeability (32); 3) Significant, prolonged bleeding (as occurs in hirudiniasis) as occurred in this dog, leading to plasma protein loss, including antithrombin (33); 4) Leech saliva contains several hirudin-like anticoagulants, directly inhibiting thrombin, potentially modulating the host's coagulation pathways (27). While these do not directly decrease ATA, they might interfere with the laboratory assay of ATA. This warrants future *in vivo* and *in vitro* studies of hemostasis after exposure to leech saliva.

Another noteworthy fact is that upon presentation, both to the primary clinic, as well as to the HUVTH-ED, is that the initial oral examinations erroneously diagnosed a 'sublingual mass', diagnosed as a hematoma, rather than the actual presence of the leeches attached to the sublingual mucosa. It was only upon a thorough oral examination, under general anesthesia, that the leeches were correctly identified. This occurrence is consistent with case reports and case series of human oral hirudiniasis (5,7, 34-37). This warrants increased awareness of veterinarians of hirudiniasis in dogs. Early identification and elimination of the leeches will prevent the occurrence of blood loss anemia and hypoproteinemia. Severe anemia is documented in young children with hirudiniasis, with reported hemoglobin levels ranging from 3 to 7.2 g/dL (37). In the present dog, its low body weight (4.6 kg) and the prolonged, approximately 10-day infestation, with two leeches, very likely contributed

to the development of the marked life-threatening blood loss anemia.

In conclusion, this report comprehensively describes an oral *L. nilotica* infestation in a dog that presented coughing, hemoptysis, melena, and severe life-threatening anemia, requiring blood transfusion. Several novel interesting hemostatic abnormalities were reported, likely reflecting the impact of leech saliva on hemostasis, including hyperfibrinolysis and increased D-dimer concentration. Leech infestation should be considered as a differential diagnosis for unexplained anemia in animals with potential exposure to contaminated water in leech endemic areas and should be carefully looked for in the oral cavity, as they might be missed during routine physical examination.

REFERENCES

1. Sket, B. and Trontelj, P.: Global diversity of leeches (Hirudinea) in freshwater. Hydrobiol. 595:129-137, 2008.
2. Ralte, M., Bordoloi, K. P., Lucy, E., Nama, D., Sonowal, P. J., Donin, A., Deka, C., Das, D., Sarma, K. and Konwar, B.: Management of nasal hirudiniasis in a hunting dog. J. Entomol. Zool. Stud. 8:391-393, 2020.
3. Montazeri, F., Bedayat, A., Jamali, L., Salehian, M. and Montazeri, G.: Leech endoparasitism: report of a case and review of the literature. Eur. J. Pediatr. 168:39-42, 2009.
4. Singh, M., Kumar, A., Kumar, A., Tyagi, S. P. and Wadhawan, R.: Case report: Endoscope-assisted bilateral nasal leech retrieval in a dog. Front. Vet. Sci. 9: 881581, 2022.
5. Raele, D. A., Galante, D. and Cafiero, M. A.: Oral hirudiniasis in a stray dog, first report in Italy. J. Vet. Med. Sci. 77:1315-1317, 2015.
6. Rajaei, S. M., Khorram, H., Ansari Mood, M., Mashhadi Rafie, S. and Williams, D. L.: Oral infestation with leech *Limnatis nilotica* in two mixed-breed dogs. J. Small Anim. Pract. 55:648-651, 2014.
7. Alshehabat, M. A.: Sublingual vein infestation with leech *Limnatis nilotica* in a hunting dog. Annal. Parasitol. 62:359-361, 2016.
8. Waner, T. and Yagil, R.: *Limnatis nilotica* infestation in a dog. Refuah Vet. 35 (1), 14, 1978.
9. Bromley, H. J.: The freshwater leeches (Annelida,) of Israel and adjacent areas, Isr. J. Zool. 40:1-24, 1994.
10. https://www.mako.co.il/news-lifestyle/2022_q3/Article-aaf042efbdb5281027.htm; Accessed on 16.08.2025.
11. Orevi, M., Eldor, A., Giguzin, I. and Rigby, M.: Jaw anatomy of the blood-sucking leeches, Hirudinea *Limnatis nilotica* and *Hirudo medicinalis*, and its relationship to their feeding habits. J. Zool. 250:121-127, 2000.
12. Machloof, M. M., Cahan, A., Edel. Y., Leibovitch, C., Ovnat-Tamir, S., Grupel, D. and Mumcuoglu, K. Y.: Hemoptysis and epistaxis caused by Nile Leach, *Limnatis nilotica*, in the hypopharyngeal wall of an adult patient in Israel. Isr. Med. Assoc. J. 26:704-706, 2024.
13. Shevkunova, E. A.: Certain data on biology of feeding of *Limnatis nilotica* (Savigny, 1820). Med. Parazitol. (Moskva) 24:346-351, 1955.
14. Negm-Eldin, M. M., Abdraba, M. A. and Benamer, H. E.: First record, population ecology and biology of the leech *Limnatis nilotica* in the Green Mountain, Libya. Travaux de l'Institut Scientifique, Rabat (Série Zoologie). 49:37-42, 2013.
15. Marschak, A.: Parasitological aspects of ENT practice in Israel. Harefuah. 54:47-48, 1958.
16. Almallah, Z.: Internal hirudiniasis in man with *Limnatis nilotica*. Iraq J. Parasitol. 54:637-638, 1968.
17. Al Safadi, M. M. and El-Shimy, N. A.: Freshwater leeches from Yemen. Hydrobiol. 263:185-190, 1993.
18. Nesemann, H. and Forster, R.: First record of *Limnatis nilotica* from Oman (Hirudinea: Hirudinidae). Miscell. Zool. Hung. 11:11-14, 1997.
19. Al-Hadrani, A., Debry, C., Faucon, F. and Fingerhut, A.: Hoarseness due to leech ingestion. J. Laryngol. Otol. 114:145-146, 2000.
20. Cheikh-Rouhou, F., Besbes, M., Makni, F., Chaabouni, M. and Ayadi, A.: (*Limnatis nilotica* causing severe anaemia in an infant. Med. Trop. (Mars) 60:100-101, 2000.
21. Agin, H., Ayhan, F. Y., Gulfidan, G., Cevik, D. and Derebasi, H.: Severe anemia due to the pharyngeal leech *Limnatis nilotica* in a child. Türk. Parazitol. Derg. 32:247-248, 2008.
22. Chen, W. C., Chien, C. Y., Yang, C.H., Li, J. H. and Hwang, C. F.: Nasal leech infestation: Report of seven leeches and literature review. Eur. Arch. Otorhinolaryngol. 267:1225-1229, 2010.
23. Solomon, E.: Leech: An unusual cause of laryngotracheal obstruction. Ethiop. Med J. 29:141-142, 1991.
24. Witenberg, G.: What is the cause of the parasitic laryngopharyngitis in the Near East ("Halzoun")? Acta. Med. Orient. (Jerusalem),3:191-192, 1944.
25. Simões, P. B., Costa, A., Santos, A. and Marcos, R.: Blood from a "half-fish" dog: how modern analyzers deal with nucleated red blood cells. Vet. Clin. Pathol. 49:8-10, 2020.
26. Taskesen, M., Katar, S. and Başçik, H.: An unusual cause of gastrointestinal bleeding and severe anemia in a child: leech infestation. J. Trop. Pediat. 55:338-339, 2009.
27. Salzet, M.: Anticoagulants and inhibitors of platelet aggregation derived from leeches. FEBS Letters. 492:187-192, 2001.
28. Chung, D. I., Moon, C. H., Kong, H. H., Choi, D. W. and Lim, D. K.: The first human case of *Clinostomum complanatum* (Trematoda: Clinostomidae) infection in Korea. Kor. J. Parasitol. 33:219-223, 1995.
29. Stokol, T., Brooks, M. B., Erb, H. N. and Mauldin, G. E.: D-dimer concentrations in healthy dogs and dogs with disseminated intravascular coagulation. Am. J. Vet. Res. 61:393-398, 2000.
30. Thachil, J., Favaloro, E. J. and Lippi, G.: D-dimers "normal" levels versus elevated levels due to a range of conditions, including "D-dimeritis," inflammation, thromboembolism, disseminated intravascular coagulation, and COVID-19. Semin. Thromb. Hemost. 48:672-679, 2022.
31. Rimpo, K., Tanaka, A., Ukai, M., Ishikawa, Y., Hirabayashi, M. and Shoyama, T.: Thrombin-antithrombin complex measurement using a point-of-care testing device for diagnosis of disseminated intravascular coagulation in dogs. PLoS One. 13:e0205511, 2018.

32. Cheng, T., Mathews, K. A., Abrams-Ogg, A. C. and Wood, R. D.: Relationship between assays of inflammation and coagulation: A novel interpretation of the canine activated clotting time. Can. J. Vet. Res. 73:97-102, 2009.
33. Kuzi, S., Segev, G., Haruvi, E. and Aroch, I.: Plasma antithrombin activity as a diagnostic and prognostic indicator in dogs: a retrospective study of 149 dogs. J. Vet. Intern. Med. 24:587-596, 2010.
34. Ağin, H., Ayhan, F. Y., Gülfidan, G., Cevik, D. and Derebaşi, H.: Severe anemia due to the pharyngeal leech *Limnatis nilotica* in a child. Türk. Parazitol. Derg. 32:247-248, 2008.
35. Cundall, D. B., Whitehead, S. M. and Hechtel, F. O. P.: Severe anaemia and death due to the pharyngeal leech *Myxobdella africana*. Transact. Royal Soc. Trop. Med. Hyg. 80:940-944, 1986.
36. Shitaye, N. and Shibabaw, S.: Severe anemia due to pharyngeal leech infestation; a case report from Ethiopia. BMC Surg. 17:102, 2017.
37. Asfaw, B. A., Yigzaw, K. T., Negussie, M. A., Gemechu, F. A., Fentaye, M. A. and Bezabih, S. A.: Unexpected culprit: High output heart failure induced by severe anemia from laryngeal leech infestation: A case report. Radiol. Case Rep. 20:4925-4928, 2025.