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CASE REPORT

Fatal pulmonary strongyloidiosis in a dog: clinical implications and first molecular identification in Chile

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Abstract. A six-month-old male Pomeranian presented persistent cough, respiratory distress, and failure to respond to empirical and palliative treatment. Diagnostic imaging revealed severe pulmonary edema. Several nematode larvae were found after microscopic analysis of bronchoalveolar lavage (BAL) performed before the patient died. PCR and sequencing identified the larvae as *Strongyloides stercoralis*.

Keywords: *Strongyloides stercoralis*; dog; pulmonary strongyloidiosis; bronchoalveolar lavage; Chile.

Strongyloides stercoralis is an intestinal parasitic helminth that mainly affects humans and dogs worldwide. It is estimated that tens of millions of people are infected worldwide. *S. stercolaris* is soil-transmitted with alternation between free-living and parasitic generations and the capacity for autoinfection, enabling chronicity and life-threatening hyperinfection in susceptible hosts (Keiser & Nutman, 2004). Infective third-stage larvae (L3) usually penetrate the skin and, during migration, pass through the lungs before reaching the small intestine. In dogs, infection may also occur via oral or trans-mammary routes (Deplazes *et al*., 2016). Human strongyloidiosis has been documented in Chile, mainly in institutionalized or immunosuppressed patients (Garibaldi *et al*., 1990; Mercado *et al*., 2002; Oddo & Duarte, 1983; Sandoval *et al*., 2004); however, data on dogs are scarce, with only indirect evidence from fecal parasitological surveys (Alcaíno & Gorman, 1999; López *et al*., 2006). In Santiago, we report a fatal case of pulmonary strongyloidiosis in a dog, with cytological and molecular confirmation from bronchoalveolar lavage (BAL) material.

An intact male six-month-old Pomeranian was presented to a veterinary hospital due to persistent cough for the last month. The patient had been purchased from a pet shop two months prior, was fully vaccinated, and received ectoparasite treatment, but no anthelmintic was administered. The dog had daily episodes of cough, serous nasal discharge, and occasional vomiting. A corticosteroid regimen was initiated one week prior to the referral. The dog shared an apartment with a 4-month-old dog that did not display any symptoms.

At the initial physical examination, the physiological constants were within the normal range, with a positive tracheal reflex. The dog was initially treated with Prednisolone Acetate (1 mg/kg/day), with the dose subsequently increased to 9 mg/day. In addition, salmeterol (25 µg) and fluticasona propionate (250 µg) were administered, along with a combination of chlorpheniramine (0.04 g/100 mL), codeine (0.2 g/100 mL) and pseudoephedrine (0.6 g/100 mL). Tracheoscopy revealed normal secretions in the principal bronchi and intrathoracic trachea flattening with a D-shape consistent with tracheal collapse grade 1. CBC showed leukocytosis (33,500/µL; RI 6,000–17,000) with neutrophilia (26,130/µL), left shift (bands 1,005/µL) and monocytosis (1,675/µL). Serum biochemistry revealed mild increases in ALP and urea levels.

Fourteen days later, the dog returned with clinical deterioration, showing persistent cough, depression, anorexia, and diarrhea for three days. The findings included cyanotic mucous membranes, capillary refill time of 2s, tachypnea, nasal obstruction, and 92% oxygen saturation. Thoracic ultrasound revealed severe pulmonary edema. Bronchoalveolar lavage (BAL) was performed and submitted to a reference laboratory for analysis. Hematology showed anemia (HCT 29.9%; RI 40–60%), hypoproteinemia (5.4 g/dL; RI 5.5–7.5 g/dL), and hypocalcemia. The dog required mechanical ventilation. The treatment comprised dexamethasone (0.5 mg/kg bid), omeprazole (0.7 mg/kg bid), meropenem (15 mg/kg bid), amikacin (20 mg/kg bid), ceftriaxone (22 mg/kg q8h), and continuous infusions of 0.9% NaCl (3 mL/kg/h) with ketamine (3 mg/kg/h), fentanyl (7 µg/kg/h), midazolam (0.5 mg/kg/h), and norepinephrine

(0.1 mg in 1 mL at 1 mL/h); rocuronium 0.2 mg/kg IV boluses were added. Hematocrit declined to 15%, and blood transfusion increased to 28%. With a blood pH of 7.0 and no clinical improvement, euthanasia was elected before BAL results were available.

Cytological examination of the BAL smears revealed chronic suppurative inflammation, with numerous nematode larvae measuring approximately 240 µm in length and 12-20 µm in width (Figure 1 A and B). DNA was isolated from the cytological slides using the EZNA® Tissue DNA Kit (Omega Bio-Tek). DNA was used in two PCRs using Taq polymerase (GoTaq G2 Green Master Mlx, Promega). Initial PCR was performed to amplify a section of the cox1 gene using the universal primers LCO1490 and HCO2198 (Folmer *et al*., 1994). Sanger sequencing yielded a 538-nt read (accession number: PZ017152) with 100% identity to *S. stercoralis* GenBank entries (NC_028624 and AJ558163). A second PCR with S. stercoralis–specific primers SSC-F/SSC-R (Hai *et al*., 2022) (accession number: PZ018879) matched a repetitive element of *S. stercoralis* with 98.95% identity (LL999473), confirming the diagnosis from the BAL.

negative (Kirkwood & Šlapeta, 2024). Collectively, these reports underscore the broad clinical spectrum of disease in dogs and the potential for life-threatening disease in young or immunocompromised animals and those receiving corticosteroids.

Diagnosis in dogs is notoriously challenging because larval shedding can be scant and intermittent. Baermann examination remains the fecal test of choice but is not routinely performed in many clinical laboratories; repeated sampling is often required. In some veterinary studies, necropsy or molecular methods confirmed infections in Baermann-negative dogs. Our case highlights a less common diagnostic route: direct cytological visualization of larvae in BAL during severe respiratory compromise, with molecular confirmation from slide-extracted DNA. When respiratory signs are prominent, BAL cytology plus PCR can be decisive and should be considered alongside serial Baermann tests and fecal PCR.

From a One Health perspective, the detection of *S. stercoralis* in dogs in Chile is epidemiologically relevant. Human cases, sporadic and often in high-risk settings, have

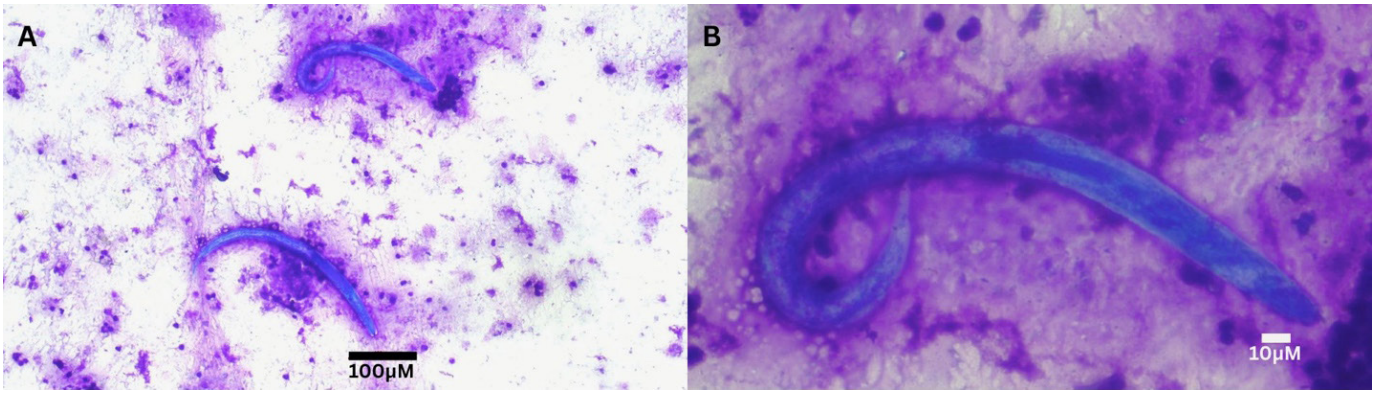

Figure 1.
Strongyloides stercoralis larvae in bronchoalveolar lavage (BAL) cytology from a six-month-old dog with fatal pulmonary strongyloidiosis. (A) Low-power field showing larvae within a neutrophilic, proteinaceous background. (B) Higher-magnification view of one of the larvae.

To our knowledge, this case represents the first molecularly confirmed case of canine pulmonary strongyloidiosis in Chile. The natural history—young age, lack of prior anthelmintic treatment, and recent corticosteroid use—likely favored larval proliferation and migration. Comparable severe outcomes have been described in Europe: a Boston Terrier with chronic gastrointestinal disease in Romania developed hyperinfection but recovered after prolonged therapy (Deak *et al*., 2024), whereas a disseminated case in the Czech Republic was fatal despite treatment (Nosková *et al*., 2024). In contrast, milder or subclinical infections were reported in Argentina, diagnosed using the Baermann technique and confirmed by PCR (Borrás *et al*., 2023), and in temperate Australia, where one asymptomatic dog was PCR-positive while routine tests were

been documented in Chile (Oddo & Duarte, 1983; Garibaldi *et al*., 1990; Mercado *et al*., 2002; Sandoval *et al*., 2004). The canine reservoir, environmental contamination with L1 larvae that can mature to infective L3 in suitable conditions, and close human–pet contact in urban apartments argue for heightened vigilance. Pet shop acquisition, lack of prior anthelmintic treatment, and subsequent corticosteroid therapy were plausible contributors in this case. A limitation of this report is that we were unable to access the cohabiting dog for clinical evaluation or sampling, precluding the assessment of within-household transmission. We recommend that Chilean veterinarians: i) consider *S. stercoralis* in coughing puppies with lower-airway disease, especially if corticosteroids have been used; ii) request fecal Baermann (serial) and/or PCR when clinical suspicion

exists; iii) institute evidence-based anthelmintic therapy (e.g., macrocyclic lactones or benzimidazoles in repeated/combined regimens per current guidelines) and avoid corticosteroids until parasitism is excluded; and iv) implement environmental hygiene and household screening, particularly for cohabiting young or immunocompromised animals and humans. Establishing local diagnostic capacity (Baermann, concentration methods, and PCR) and surveillance in dogs would clarify the zoonotic ecology of *S. stercoralis* in Chile.

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