Title

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Journal

Journal of Veterinary Internal Medicine, 35(3)

ISSN

0891-6640

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Publication Date

2021-05-01

DOI

10.1111/jvim.16110

Peer reviewed

Received: 1 November 2020 | Accepted: 19 March 2021

DOI: 10.1111/jvim.16110

CASE REPORT

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

Lymphoplasmacytic and eosinophilic enteritis with or without globule leukocyte hyperplasia in 4 goats

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Abstract

Four, mature, client-owned goats were presented to referral hospitals for recurrent diarrhea despite treatment for intestinal parasitism. Common clinical findings included diarrhea, poor condition, neutrophilia, and hypoalbuminemia. Testing for common infectious causes of diarrhea in goats was negative. Ultrasonography and computed tomography in 2 cases was suggestive of enteritis, including thickened intestinal walls and fluid filled, dilated small intestines, respectively. Lymphoplasmacytic and eosinophilic enteritis (LEE) was ultimately diagnosed on intestinal biopsy histopathology based on the presence of small intestinal villous blunting and increased numbers of lymphocytes and eosinophils predominantly within the lamina propria. Numerous globule leukocytes were also noted on histopathology in 3 cases. All goats responded favorably to corticosteroid treatment with weight gain and resolution of diarrhea and clinicopathologic abnormalities. Relapses occurred, and complete cure was difficult to achieve. Reported in other species, this series describes the clinical presentation, diagnosis, and treatment of LEE in adult goats.

KEYWORDS
caprine, hypoalbuminemia, inflammatory bowel disease, weight loss

1 | INTRODUCTION

Lymphoplasmacytic and eosinophilic enteritis is characterized by intestinal infiltration with lymphocytes, plasma cells, and eosinophils. The mucosal and submucosal layers can be affected along any part of the gastrointestinal tract.[1,2] Inflammatory bowel diseases with eosinophilic infiltrates can be idiopathic or secondary to parasitism, drug reaction, systemic eosinophilic syndrome, or malignancy in ruminants and other species.[3-5] In cases without an identified underlying cause, immunosuppressive therapy with corticosteroids is often pursued.[3] Clinical presentation varies depending on the gastrointestinal region affected, but includes impaired digestion and absorption resulting in diarrhea and ill-thrift with hypoproteinemia,[3,5-8] signs commonly associated with endoparasitism.[9] Numerous globule leukocytes (GL) occur in the small intestine of sheep with nematode infestation,[10,11] and are present in intestinal mucosa and involved in mucosal immunity in mice.[12] Primary LEE with or without a GL component has not been reported in small ruminants. This report describes the presentation, diagnosis, and treatment of LEE in 4 goats.

Abbreviations: AMDUCA, Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994; BCS, body condition score; CL, *Corynebacterium pseudotuberculosis*; GL, globule leukocytes; IBD, inflammatory bowel disease; LEE, lymphoplasmacytic and eosinophilic enteritis; MAP, *Mycobacterium avium* subspecies *paratuberculosis*.

2 | MATERIALS AND METHODS

A retrospective review of medical records was conducted. Inclusion criteria included histopathologic diagnosis of LEE with or without GL hyperplasia on intestinal biopsies. Signalment, clinical history, physical examination findings at presentation, results of diagnostic procedures, and treatment were recorded when available.

3 | RESULTS

3.1 | Case 1

A 2-year-old, Nigerian Dwarf buck, presented for a 5-month history of intermittent diarrhea (Supplementary Table 1). The buck's ration consisted of grass and alfalfa hay. Treatment prior to presentation included multiple, oral medications including several 5-day courses of sulfadimethoxine; a single dose of ivermectin; and multiple doses of kaopectate bismuth, toltrazuril, and psyllium, without response. Fecal flotation, fecal culture, and serologic testing for *Corynebacterium pseudotuberculosis* (CL), *Mycobacterium avium* subspecies *paratuberculosis* (MAP), and lentiviruses prior to presentation were negative.

On presentation, the buck was bright, alert, responsive, afebrile (temperature 102.6 °F; ref: 101.5-104 °F), tachycardic (120 bpm; ref: 70-90) and tachypneic (50 bpm; ref: 10-30).[13] Subjective, pear-shaped abdominal distension was evident, with normal rumen contractions and pelleted manure. No peripheral lymphadenopathy was appreciated.

Clinicopathologic abnormalities included normocytic, normochromic, regenerative anemia (PCV 22.8%; ref: 23-36%; reticulocytes 190 560/μL; ref: rare), leukocytosis (19 760/μL; ref: 5000-17 000/μL) characterized by mature neutrophilia (17 389/μL; ref: 700-7600/μL), with hyperglycemia (156 mg/dL; ref: 45-70) and hypoalbuminemia (2.3 g/dL; ref: 3.3-4.2). A fecal PCR test for MAP was negative. Plasma trace element screen revealed a hypoferremia (0.89 ppm; ref: 1.5-2.5). Fecal McMaster test had 200 oocysts/g of *Eimeria*. Abdominal ultrasound was unremarkable.

Exploratory laparotomy was pursued for additional gastrointestinal evaluation and to obtain intestinal biopsies since no definitive cause of chronic diarrhea had been identified. Full-thickness biopsies of the duodenum, jejunum, and cecum identified chronic, diffuse, lymphoplasmacytic, and eosinophilic inflammation predominantly of lamina propria with numerous intraepithelial GL and small intestinal villous blunting (Figure 1). A single, enlarged mesenteric lymph node had sinus histiocytosis consistent with a nonspecific draining reaction.

The buck was diagnosed with LEE with GL hyperplasia and treated with prednisolone (1 mg/kg PO q12h) for 3 weeks, based on the reported treatment of eosinophilic enteritis in cattle,[5] before reducing the dose by approximately 30% every other week for 6 weeks. A strict grass and oat hay diet was instituted in case intestinal inflammation represented a possible dietary allergic reaction, as described in humans and cats with inflammatory bowel disorders.[3,6,14] Six weeks after discharge, the buck had gained 3.2 kg and had improved manure consistency and serum albumin (3.1 g/dL).

3.2 | Case 2

A 6-year-old, Nigerian Dwarf doe, presented with a 4-month history of intermittent diarrhea (Supplementary Table 1). The doe was housed with another similarly aged, healthy doe. Ration consisted of pasture and grass hay, with occasional alfalfa and grain. Treatment before presentation included a 5-day course of sulfadimethoxine and a single dose of ivermectin PO after which diarrhea resolved for 1 month. At that time, quantitative fecal flotation and McMaster test found a low number (100 oocysts/g) of *Eimeria* spp. Access to lush pasture was restricted; however, the doe became lethargic and lost weight as diarrhea progressed and was presented for evaluation.

At presentation, the doe was bright, alert, responsive, and afebrile (temperature 102.7 °F) with normal heart rate (72 bpm), bruxism, and

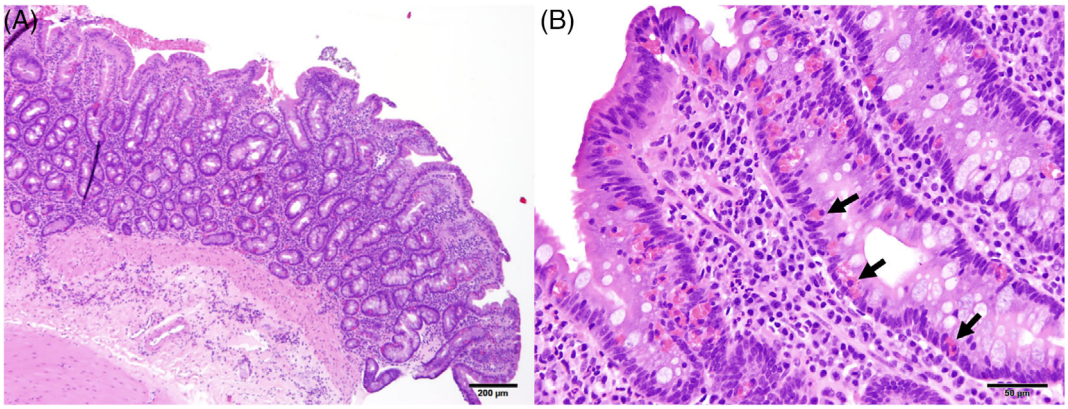

FIGURE 1 Histopathology of intestinal biopsies from case 1 with . lymphoplasmacytic and eosinophilic enteritis. A, The lamina propria of the duodenum is expanded by a dense inflammatory infiltrate that variably extends into the submucosa. Villi are diffusely blunted and often fused. H&E, ×4. B, The duodenal mucosa is infiltrated with abundant lymphocytes, plasma cells, and eosinophils. Numerous globular leukocytes (arrows) are located within the mucosal epithelium. H&E, ×20

tachypnea (54 bpm). Body condition score (BCS) was 2.5 of 5. Rumen contractions (1/min) were strong. Diarrhea was passed during examination. No peripheral lymphadenopathy was appreciated.

Clinicopathologic abnormalities included mature neutrophilia (9412/μL; ref: 700-7600/μL), with hyperglycemia (131 mg/dL; ref: 45-70), hypoalbuminemia (2.9 g/dL; ref: 3.3-4.2), and hypocalcemia (7.9 mg/dL; ref: 8.9-11.2). Fecal PCR and serum antibody ELISA for MAP were negative. Serum hemolysis inhibition for CL was also negative, as was fecal rotavirus ELISA. Repeated *Salmonella* cultures (6) were negative. Plasma trace element screen was unremarkable. Abdominal ultrasound revealed small intestinal thickening (0.32-0.46 cm; ref: 0.08-0.21)[15,16] with associated mesenteric lymphadenopathy (Figure 2).

An exploratory laparotomy was performed, and biopsies of the jejunum and ileum identified diffuse, chronic lymphoplasmacytic, and eosinophilic enteritis infiltrating predominantly the lamina propria with mucosal edema, submucosal lymphoid hyperplasia, and intraepithelial GL. Punch biopsy of a mesenteric lymph node had sinus histiocytosis consistent with a nonspecific draining reaction.

The doe was diagnosed with LEE with GL hyperplasia and administered dexamethasone (.2 mg/kg SC once) followed by prednisone (1 mg/kg PO q12h) for 3 weeks, based on reported treatment of eosinophilic enteritis in cattle,[5] before reducing the dose by 50% every 4 days for a total of 8 days. One month later, the manure consistency and small intestinal thickening (0.29 cm) had improved. The inflammatory leukogram and hypoalbuminemia (3.6 g/dL) had resolved. Over the next 2 years, relapses occurred intermittently with diarrhea, hypoalbuminemia (2.8 g/dL and 2.3 g/dL), and ultrasonographically visible, subjectively thickened small intestine whenever corticosteroid medications were discontinued. Signs recurred after 1- and 2-month discontinuation of corticosteroids, and while receiving a consistent low dose (0.1 mg/kg PO q48h). The doe responded initially to an increased dose of prednisone (0.2 mg/kg PO q12h) followed by a longer taper, decreasing the dose weekly by 50% for 4 weeks. Due to recurrent signs, the doe was maintained over time on prednisone (3 mg/kg PO q24h) with intermittent, concurrent cetirizine (0.2 mg/kg PO q12h) because of its inhibition of eosinophil survival.[17] On re-evaluation, the doe's manure had normalized and BCS improved.

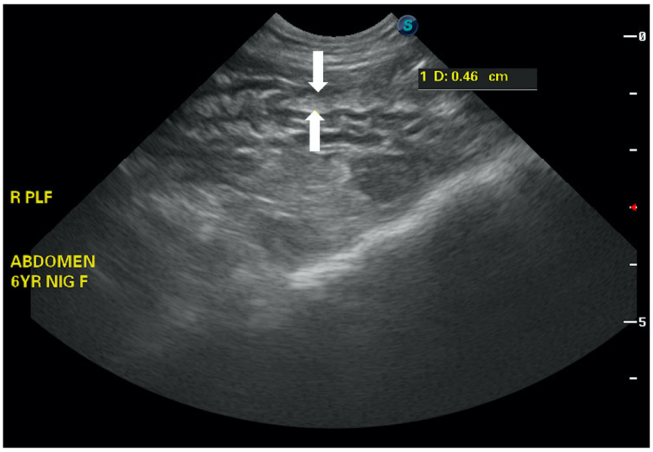

FIGURE 2 Transabdominal ultrasonographic image of case 2 obtained from the right paralumbar fossa (R PLF) of a thickened (0.46 cm) small intestinal wall segment (arrows). Image was obtained at 5.5 to 8.5 MHz with a microconvex curvilinear transducer at a depth of 5.9 cm

3.3 | Case 3

An 18-month-old, Anglo Nubian doe, presented for an approximately 1-year history of intermittent diarrhea (Supplementary Table 1). The doe was the only goat on the premises and shared a pasture with horses. Diagnostics performed before presentation included fecal flotation, which identified a coccidia load (unknown amount), and MAP testing (unknown test), which was negative. Oral treatment with sulfadimethoxine, ponazuril and amprolium (unknown doses and duration) before presentation did not improve the diarrhea although the coccidiosis resolved on subsequent repeat fecal flotations.

At presentation, clinicopathologic abnormalities included leukocytosis (23 000/μL; ref: 5000-17 000/μL) characterized by eosinophilia (5980/μL; ref: 50-650/μL) and mature neutrophilia (9660/μL; ref: 700-7600/μL) with hypoalbuminemia (2.1 g/dL; ref: 3.3-4.2). Initial management consisted of discontinuing current medications (sulfadimethoxine, ponazuril, and amprolium) and limiting feed to grass hay and a commercial goat feed to rule out possible allergic responses contributing to the eosinophilia. On re-evaluation 1 month later, eosinophilia resolved (480/μL), though diarrhea persisted. After another month of dietary restriction, the doe developed submandibular edema, ascites and normocytic, normochromic, nonregenerative anemia (PCV: 12%). A whole blood transfusion was performed. Despite initial improvement in attitude and appetite after transfusion, diarrhea recurred 4 days later.

An exploratory laparotomy was performed to investigate chronic diarrhea that could not be attributed to intestinal nematodiasis. Jejunal, ileal, and cecal biopsies identified diffuse eosinophilic, lymphoplasmacytic, and histiocytic inflammation predominantly of lamina propria with numerous intraepithelial GL, small intestinal villous blunting, and a focal jejunal granuloma. The doe was diagnosed with LEE with GL hyperplasia and treated with dexamethasone (0.05 mg/kg SC q24h) for 1 month, based on reported corticosteroid dosage in horses,[18] before increasing the interval between doses to every other day for 5 doses, then every 2 days for 5 doses and finally once weekly for 5 doses before discontinuing. The doe responded well and 1 year after discharge she remained clinically healthy, and still receiving dexamethasone (0.05 mg/kg SC once weekly).

3.4 | Case 4

A 7-year-old Nigerian Dwarf buck presented for a 3-year history of weight loss without inappetance and a 6-month history of intermittent fever and diarrhea (Supplementary Table 1). Other goats on the

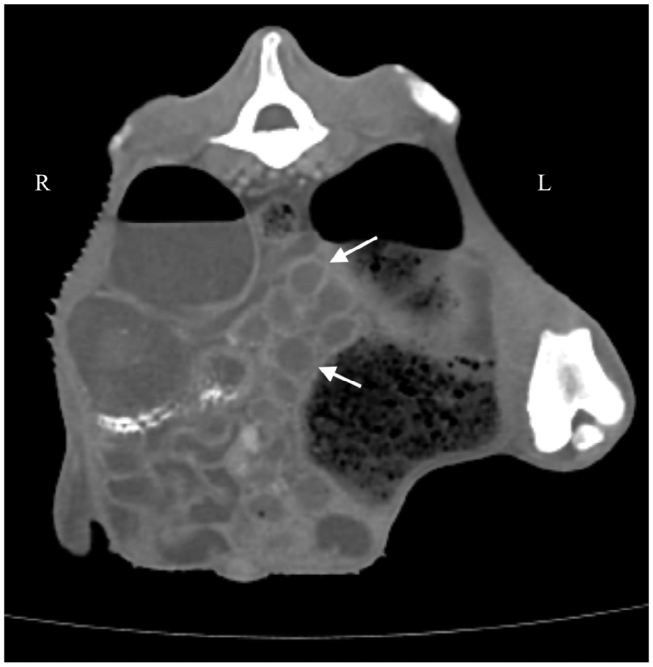

FIGURE 3 Soft-tissue window, transverse computed tomography image obtained after intravenous iodinated contrast injection, of the abdomen of case 4. "R" indicates the right side of the animal. Arrows indicate fluid-filled small intestinal loops

property (varying ages and production status) were apparently healthy. Routine herd management included deworming with a combination ivermectin and clorsulon product guided by McMasters fecal egg counts. Before presentation, the buck was treated for intestinal parasitism; louse infestation and fever with ceftiofur, flunixin meglumine, toltrazuril, ivermectin, and clorsulon; topical permethrin insecticide; and a vitamin supplement. Despite initial improvement, diarrhea recurred when treatment was discontinued.

On presentation, the buck was bright, alert, responsive, afebrile (temperature 101.6 °F), eupneic (24 bpm), and tachycardic (96 bpm). He weighed 25.9 kg with BCS 1 of 5. Mucous membranes were pale. Rumen contractions (2/min) were quiet. No lameness or neurologic deficits were noted; however, the buck remained recumbent. There was no external lymphadenopathy.

At presentation, clinicopathologic abnormalities included normocytic, normochromic, nonregenerative anemia (PCV 18%; ref: 22-38%), and leukocytosis (43 160/μL; ref: 4000-13 000/μL) characterized by mature neutrophilia (35 391/μL; ref: 1200-7200/μL) and monocytosis (863/μL; ref: 0-550/μL), with hypoalbuminemia (2.0 g/dL; ref: 2.3-4.0), hypocalcemia (6.3 mg/dL; ref: 9-12), hypoglycemia (44 mg/dL; ref: 50-75), and global increase of liver enzymes (AST: 140 IU/L, ref. 30-80; GGT: 78 IU/L, ref. 5-52; SDH: 78 IU/L, ref. 14-24). Fecal McMaster revealed trichostrongyles (725 ova/g). Fecal PCR and serum antibody ELISA for MAP were negative. Aerobic fecal culture was unremarkable. Computed tomography of the abdomen was performed for comprehensive assessment of chronic gastrointestinal disease nonresponsive to preceding treatment measures. Abnormal findings included diffusely fluid-filled small intestines (Figure 3).

Full-thickness biopsies of the jejunum and cecum obtained at exploratory laparotomy identified eosinophilic and lymphoplasmacytic enteritis and typhlitis, respectively, affecting predominantly the lamina propria with small intestinal villous blunting, intraepithelial nematode larvae (consistent with *Trichostrongylus* spp.), and coccidial organisms. Histopathology of an enlarged lymph node had lymphoid reactivity with suppurative inflammation and multifocal parasitic granulomas.

Based on these findings, the buck was diagnosed with LEE with encysted parasites and treated with dexamethasone (0.1 mg/kg SC once) followed by prednisone (1.1 mg/kg PO q12h) depending on the reported treatment of eosinophilic enteritis in cattle.[5] Parasites remained a treatment challenge, with evidence of resistance to moxidectin (2 weeks after moxidectin treatment the *Trichostrongylus* egg count decreased by 75%). Regular fecal flotation guided continued antiparasitic therapy, which included levamisole and toltrazuril. The buck was discharged on a 2-month course of prednisone after which the dose was decreased weekly by 15% before being discontinued. Nine months later, the buck had gained 16 kg and clinicopathologic abnormalities resolved.

4 | DISCUSSION

This case series describes the presentation, diagnosis, and treatment of LEE in goats. The 4 cases described in this report identify several common clinical findings in goats with LEE, including chronic, intermittent diarrhea nonresponsive to antiparasitic treatment, weight loss, hypoalbuminemia, neutrophilia, and anemia. These clinical signs are nonspecific, and therefore, LEE is a diagnosis of exclusion confirmed by histopathology. All goats were diagnosed with LEE with or without GL hyperplasia based on clinicopathologic and imaging findings, exclusion of MAP and substantial parasitism as the cause of disease, and ultimately, characteristic intestinal histopathology.

Intestinal parasitism was diagnosed historically (case 3) or at the time of presentation (case 4) in 2 goats, possibly contributing to the intestinal inflammation in these patients. Cases 1 and 2, however, did not have evidence of intestinal parasitism and had a history of periodic anthelmintics. Lack of endoparasitism at presentation, however, does not rule out previous endoparasitism as a possible initiator of the enteritis. Cases 1, 2, and 3, with no evidence of endoparasitism at the time of biopsy acquisition, had GL hyperplasia while case 4, with a parasite load at the time of biopsy acquisition, lacked GL. The presence of GL may indicate prior endoparasitism even in cases with negative fecal flotations. Also referred to as intraepithelial mast cells of the intestine, GL are considered important for mucosal immunity and have been associated with endoparasitism in ruminants.[11,12] Increased intraepithelial GL have been associated with rapid immune expulsion of nematode larvae as well as breed-related resistance to nematodiasis in sheep.[19,20] These findings suggest a relationship between endoparasitism and diagnosis of LEE in all cases reported here.

The cause of the anemia in cases 1, 3, and 4 was attributed to anemia of inflammatory disease, with likely contribution from the

endoparasite burden in case 4.[21] Due to the profound leukocytic response in case 4, crowding of the bone marrow with subsequent reduction in hematopoiesis may have occurred and was supported by the lack of overt evidence of a regenerative response such as polychromasia or presence of nucleated red blood cells.

Ultimately, immunosuppressive medications such as corticosteroids are often essential in the treatment of chronic enteropathies of unknown cause.[3] Specific drug and dosing regimens for treatment of LEE in goats are unavailable and certain concerns exist in regard to bioavailability and conversion of prednisone into the active form of prednisolone, as reported in other species.[22-24] In addition, oral drug administration in ruminants raises concerns for drug inactivation within the rumen and poor absorption.[25,26] All cases in this series responded favorably to 1 to 2 months of primarily oral corticosteroid therapy with normalization of manure and improved serum albumin concentrations (when available). Evidence of recurrence of disease in case 2, however, emphasizes the need for continued monitoring in these cases and potentially chronic, long-term treatment with possible disease progression. As corticosteroid medications have no label coverage for ruminant species, and no definitive information regarding appropriate withdrawal times, owners should be advised in advance of their use and the most current information available regarding withdrawals for these medications should be determined by communicating directly with Food Animal Residue Avoidance Databank.[27] Furthermore, immunosuppressive treatment of LEE complicates management of parasite burdens in treated animals, potentially highlighting the need to treat endoparasitism before instituting corticosteroid treatment and parasite monitoring via regular fecal flotation.

Independent of the initiating cause, inflammation of the intestinal mucosa, lamina propria, and submucosa explains the partial intestinal thickening and decreased function resulting in malabsorption and diarrhea seen in these goats.[7,8,28] This has been well documented in cats and people.[3,14] The clinical presentation of goats with LEE is similar to reports in other species with eosinophilic enteritis including cattle, cats, and dogs.[4-7,14] Weight loss is inconsistent in cattle, as it was in the goats, and might relate to the duration or severity of disease.[5] Interestingly, hypoalbuminemia is not a major feature of this disease in cattle or cats.[4,5,14]

In conclusion, LEE should be included as a differential diagnosis in adult goats with chronic, intermittent diarrhea and hypoalbuminemia, with or without weight loss. The etiology of LEE is uncertain. Moreover, the role of intestinal parasitism in establishing intestinal inflammation and the significance of numerous GL warrant further investigation. This disease is responsive to a range of common corticosteroid agents, resulting in effective short-term management of clinical signs.

ACKNOWLEDGMENT

No funding was received for this study. Dr. Stieger-Vanegas provided the computed tomography image.

CONFLICT OF INTEREST DECLARATION

Authors declare no conflict of interest.

OFF-LABEL ANTIMICROBIAL DECLARATION

Ceftiofur and sulfadimethoxine were used in an off-label manner, in accordance with AMDUCA regulations for minor food animal species.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Consent was gained from the owners for all diagnostic procedures and use of case material for teaching and research purposes.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

How to cite this article: Pitel MO, Heller MC, Roberson JR, McKenzie EC, Sinnott DM, Affolter VK. Lymphoplasmacytic and eosinophilic enteritis with or without globule leukocyte hyperplasia in 4 goats. *J Vet Intern Med*. 2021;35:1620–1625. https://doi.org/10.1111/jvim.16110