Diagnosis of systemic lupus erythematosus in a dog with an atypical presentation of pleuritis

Received: 9 August 2023 | Revised: 27 September 2023 | Accepted: 29 September 2023

DOI: 10.1002/vrc2.747

Vet Record CaseReports

CASE REPORT

Companion or pet animals

Diagnosis of systemic lupus erythematosus in a dog with an atypical presentation of pleuritis

Valerie S. W. Oh[1,2] | **Maverick Melega**[1] | **Suzanne Bussey**[1] | **Craig R. Breheny**[1] | **Daria Starybrat**[1]

[1]The Royal (Dick) School of Veterinary Studies, University of Edinburgh, Midlothian, UK

[2]Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California-Davis, Davis, California, USA

Correspondence
Valerie S. W. Oh, Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California-Davis, Davis, CA, USA.
Email: valerieoh0224@gmail.com

Abstract

A 3-year-old labradoodle presented with prior history of generalised seizures and had recently developed acute pyrexia and shifting leg lameness, with joint effusions in several joints. The dog also later developed pleuritis with an exudative pleural effusion. Cytological findings for all joint effusions were consistent with immune-mediated polyarthritis. Suspected lupus erythematosus cells were seen in the joints and pleural fluid, which were confirmed with Feulgen staining. Together with a positive antinuclear antibody titre, the dog was diagnosed with systemic lupus erythematosus, which responded to immunosuppressive doses of prednisolone. This case report describes the insidious nature of systemic lupus erythematosus, a rare disease that can imitate many different conditions. It is also the first reported case of lupus erythematosus cells in pleural effusion in veterinary medicine, highlighting the importance of lupus erythematosus cells as a valuable diagnostic aid to further support the diagnosis of systemic lupus erythematosus and enable early treatment to be initiated.

KEYWORDS

cytology, dogs, immune-mediated diseases, internal medicine

BACKGROUND

Systemic lupus erythematosus (SLE) is a rare multisystemic autoimmune syndrome, in which there is a loss of self-tolerance, causing inappropriate lymphocyte activation. This results in the production of autoantibodies that target various cellular components, leading to the formation of immune-mediated complexes in the absence of an infection or other discernible cause.[1] Depending on the site of deposition of these circulating immune complexes in various organs or tissues, they can induce tissue inflammation and damage, leading to a variety of clinical signs attributable to numerous body systems.[2] The clinical syndrome is characterised by inflammation involving several body systems, including polyarthritis, anaemia, thrombocytopenia, glomerulonephritis, neurological signs and serositis.[3–6]

The underlying aetiology and mechanism of the pathogenesis remain unclear. The American College of Rheumatology (ACR) introduced a classification system with 11 criteria included to help diagnose SLE.[6] There is no equivalent classification system in veterinary medicine, and hence, a modified classification modelled against this existing human system was proposed by Ettinger et al. for use in dogs (Table 1).[1] While a positive antinuclear antibody (ANA) titre is often associated with this condition, it lacks specificity and can also be present in several other non-immune-mediated diseases, such as ehrlichiosis.[7] Another diagnostic method that supports a diagnosis of SLE is the presence of lupus erythematosus (LE) cells, which are phagocytes, mainly neutrophils, engulfing opsonised nuclear material.[8] They can be demonstrated in vitro or in vivo, and Feulgen reaction is used to highlight the DNA content. Even though LE cells are only rarely identified in vivo, they are highly suggestive of SLE.[9] In people, they have been described in synovial fluid, body cavity effusions, bone marrow, peripheral blood, bronchoalveolar lavage (BAL) and cerebrospinal fluid.[8,10,11] Similarly, LE cells are an uncommon finding in domestic species; they are rarely observed in synovial fluid,[5] and have been reported in the BAL of a dog.[12]

While the diagnosis of SLE remains challenging due to its non-specific manifestation, this case report illustrates the insidious nature of SLE as 'the great imitator' of other diseases. We also report for the first time the presence of LE cells in the pleural fluid of a dog, which highlights the importance of

careful cytological evaluation of serous fluid to provide clues for the diagnosis.

CASE PRESENTATION

A 29 kg, 3-year-old, neutered, male labradoodle was presented with pyrexia (40°C), ataxia, lethargy, hyporexia and stiff gait. The dog had a previous history of facial dermatitis, suspected to be due to skin allergies and sneezing episodes that were managed with anti-inflammatory doses of glucocorticoids for more than 6 months. Glucocorticoids had been discontinued 3 weeks before presentation. The dog also had a history of generalised tonic-clonic seizures, occurring once to twice a month for 7 months before referral, which had then progressed to cluster seizures. All seizure episodes lasted between 1 and 2 minutes. At the time, investigations with complete blood count, serum biochemistry and neurological examination at primary care veterinarian were unremarkable. The dog was treated with imepitoin (Pexion, Boehringer Ingelheim), which did not improve the seizure episodes. The dog was subsequently referred to the neurology service for further investigations. The owners opted against advanced imaging, and elected to trial medical treatment with phenobarbital (3 mg/kg, orally [PO] twice daily [BID]; Epityl, Chanelle Pharma) based on neurological examination alone and a presumptive diagnosis of idiopathic epilepsy. The dog then represented 1 month later, after developing a waxing and waning pyrexia, new onset heart murmur, shifting lameness and ulceration on the tongue with associated halitosis.

On presentation, the dog was obtunded with a body condition score of 4/9. A grade II/VI right-sided, holosystolic heart murmur was auscultated over the apex. The heart rhythm was regular with synchronous pulses. There were no adventitious lung sounds appreciated. There was generalised peripheral lymphadenopathy. The dog was ambulatory with marked stiffness and discomfort on manipulation of the joints, with palpable joint effusions present in both tarsi. Neurological evaluation confirmed the stiff gait with low head carriage and ataxia. The remainder of the neurological examination was within normal limits. The rectal temperature was 38.3°C on presentation, but fluctuated between 38.1°C and 40.2°C during hospitalisation, with the stiffness progressively worsening.

INVESTIGATIONS

Haematology revealed a mild normochromic normocytic non-regenerative anaemia (haemocrit 36%; reference interval [RI]: 39%–55%) suspected to be due to chronic inflammation, and mild thrombocytopenia that was confirmed on the blood smear evaluation (98 K/μL; RI: 148–484 K/μL). Leukogram revealed a normal neutrophil (3.21 $\times$ 10^9/L; RI: 2.95–11.64) and monocyte counts (0.78 $\times$ 10^9/L; RI: 0.16–1.12), with mild lymphopenia (1.31 $\times$ 10^9/L; RI: 1.95–5.10), indicative of a glucocorticoid (stress, steroid therapy) response. Serum biochemistry demonstrated a mild hypoproteinaemia (46.8 g/L; RI: 58–73 g/L) due to a moderate hypoalbuminaemia (18.3 g/L; RI: 26–35 g/L), likely due to a combination of inflammation, and possible loss via the gastrointestinal tract. There was a markedly elevated C-reactive protein (91.9 mg/L; RI: 0–5 mg/L) indicative of inflammation, and the rest of the values were otherwise within normal limits. Antibody detection of *Ehrlichia canis, Anaplasma* sp., *Borrelia burgdorferi* and antigen detection for *Dirofilaria immitis* were negative (SNAP 4Dx, IDEXX). In-house urine analysis indicated inactive sediment with normal values (Urine Analysis Automated, IDEXX), and a urine sample obtained via cystocentesis did not identify any bacterial growth.

LEARNING POINTS/TAKE-HOME MESSAGES

- Diagnosing systemic lupus erythematosus is challenging due to its variable presentations. Hence, a thorough evaluation of the history, clinical signs and a positive antinuclear antibody test are required to improve the certainty of a diagnosis of systemic lupus erythematosus.
- This case report documents lupus pleuritis in canine systemic lupus erythematosus with a pleural effusion, which has not been previously reported in the veterinary literature.
- Identifying lupus erythematosus cells in pleural effusion is a valuable and rapid diagnostic tool for systemic lupus erythematosus in addition to the current classification system to diagnose systemic lupus erythematosus, and should be considered in cases presenting with body cavity effusions.

Arthrocentesis was performed on both carpi, left stifle and left tarsal joints, and the slides were submitted to the clinical pathology service for evaluation. In all four joints, there were moderately increased nucleated cell counts ranging from 5.3 to 9.2 cells/40× field (normal being less than two) with a predominance of non-degenerate neutrophils (ranging from 68% to 85%, with normal limits being less than 12%). In all samples, there were occasional neutrophils and macrophages containing a differing amount of variably basophilic or pale pink to purple globular material, consistent with ragocytes (Figure 1). Rare to occasional neutrophils with one large round globular pale pink to purple cytoplasmic inclusion leading to peripheralisation of the nucleus, suspected to be LE cells (Figure 1), were also present. The presence of neutrophilic inflammation in multiple joints with ragocytes and suspected LE cells was supportive of the diagnosis of immune-mediated polyarthritis and suspected SLE. Fine-needle aspiration of the popliteal and prescapular lymph nodes identified reactive lymphoid hyperplasia indicative of antigen stimulation, likely secondary to the underlying systemic disease.

Echocardiography revealed mild thickening of the mitral valve leaflet and a moderate mitral regurgitation, alongside mild systolic dysfunction. Despite these minor changes, there was no vegetative lesion observed on echocardiography. Therefore, four blood samples were taken aseptically for *Bartonella* infectious disease testing (IDEXX Laboratories, Bartonella indirect fluorescent antibody) and additionally submitted for bacterial culture to further evaluate for endocarditis. All blood culture samples returned with no growth. Serum cardiac troponin-I levels were evaluated (i-Stat 1 Troponin, Zoetis) and yielded a normal result (<0.02 ng/mL; RI: 0.00–0.05 ng/mL).

TABLE 1 A diagnosis of SLE is established if a patient meets three or more criteria simultaneously or over a period of time.

Proposed criteria for the diagnosis of SLE	
1. Antinuclear antibody	Abnormal ANA titre in the absence of drugs, infectious diseases or neoplasia known to be associated with its development
2. Cutaneous lesions	Depigmentation, erythema, erosions, ulcerations, crusts and/or scaling, with biopsy findings consistent with SLE
3. Ocular ulcers	Oral or nasopharyngeal ulceration, usually painless
4. Arthritis	Non-erosive, non-septic arthritis involving two or more peripheral joints
5. Renal disorders	Glomerulonephritis or persistent proteinuria in the absence of urinary tract infection
6. Anaemia and/or thrombocytopenia	Haemolytic anaemia and/or thrombocytopenia in the absence of offending drugs
7. Leukopenia	Low total white blood cell count
8. Polymyositis of myocarditis	Inflammatory disease of the skeletal or cardiac muscle
9. Serositis	Presence of non-septic inflammatory cavity effusion (abdominal, pleural or pericardial)
10. Neurological disorders	Seizures, peripheral neuropathy, myopathy or cranial nerve deficits in the absence of known disorders
11. Antiphospholipid antibodies	Prolongation of activated partial thromboplastin time that fails to correct with a 1:1 mixture of patient and normal plasma, in the absence of heparin or fibrin degradation products

Note: A definite SLE diagnosis in dogs can be established if at least two major criteria with positive serology or one major criterion and two minor criteria with positive serology are present. If one major criterion with positive serology or two major criteria with negative serology are present, a diagnosis of SLE is only probable. The criteria that are met by our patient are in bold.
Abbreviations: ANA, antinuclear antibody; SLE, systemic lupus erythematosus.

Point-of-care ultrasound (POCUS) during initial triage examination revealed no abnormalities in either the thoracic or abdominal cavities. However, 48 hours following hospitalisation, the dog developed acute respiratory distress, with tachypnoea and moderately increased respiratory effort. Repeat POCUS revealed a novel bilateral pleural effusion. Full-body computed tomography (CT) of the dog was performed. CT showed a bilateral pleural effusion, multifocal lymphadenopathy and multifocal joint swelling, consistent with clinical findings from the physical examination, with no underlying cause identified.

Thoracocentesis was carried out for therapeutic purposes to relieve the respiratory distress, and a sample of effusion was submitted to the laboratory to ascertain the nature of the pleural effusion. A total of 200 mL of serosanguineous and flocculent fluid was removed, and further fluid analysis revealed a nucleated cell count of 55.9×10^9/L and a total protein of 4.0 g\dL, consistent with an exudate. Cytological examination revealed a predominant population of degenerate neutrophils often engulfing some globular material similar to ragocytes observed in the synovial fluid (Figure 1). Rare, suspected LE cells were also found and were similar in appearance to the cells identified in the joints (Figure 1).

To further confirm that the intracytoplasmic inclusions were LE cells, pre-stained slides from the left tarsus, right carpus and pleural fluid were stained with Feulgen. Briefly, May Grunwald-Giemsa-stained slides (7152 Aerospray Hematology Pro Slide Stainer, ELITech) were de-coverslipped in xylene, rinsed in distilled water, and hydrolysis was performed in a series of 1 M HCl solutions (1' RT, 8' 60°C, 1' RT). Slides were then passed in Schiff's reagent for 35 minutes, rinsed in water and counterstained with 0.02% light green in 0.2% acetic acid for 1 minute, dried and mounted. In all samples, most of the large cytoplasmic inclusions in the neutrophils and macrophages showed a bright pink-red-positive Feulgen reaction, demonstrating the presence of deoxyribose and therefore DNA in the inclusions (Figure 1).

Further evidence of SLE was achieved with antinuclear antigen antibody (ANA) titre (IDEXX Laboratories), which was highly positive with a titre of 1:1600, indicative of SLE (RI: <1:40).

DIFFERENTIAL DIAGNOSIS

The main differential diagnosis for the above clinical and diagnostic findings was SLE or infective endocarditis, with the latter deemed less likely given the combination of imaging, microbiology and biomarker evaluations.

TREATMENT

With the potential of endocarditis, antibiotic therapy (clindamycin 11 mg/kg intravenously [IV] BID, Zodon Ceva Animal Health; and marbofloxacin 5 mg/kg IV once daily [SID], Marbocyl Vetoquinol UK) was commenced while diagnostics were pursued. The decision to initiate immunosuppressive doses of glucocorticoid therapy (dexamethasone 7.1 mg/m^2, 0.953 BSA, IV SID; Colvasone, Norbrook) was made after receiving the cytology results, which showed the presence of LE cells in the pleural and synovial effusions. This decision was further supported by echocardiographic findings and normal serum troponin, although the sensitivity and specificity of this latter diagnostic limited the clinical utility.[4] Antibiotic therapy was discontinued once the negative blood culture results were received.

OUTCOME AND FOLLOW-UP

Forty-eight hours after commencing glucocorticoid therapy, the patient showed significant clinical improvement with resolution of pyrexia, mouth ulceration and pain. There was also marked improvement in gait and the joint effusions. The pleural effusion was scant on repeat POCUS. The patient was then discharged on anti-seizure medication in the form of phenobarbitone (3 mg/kg PO BID; Epityl, Chanelle Pharma),

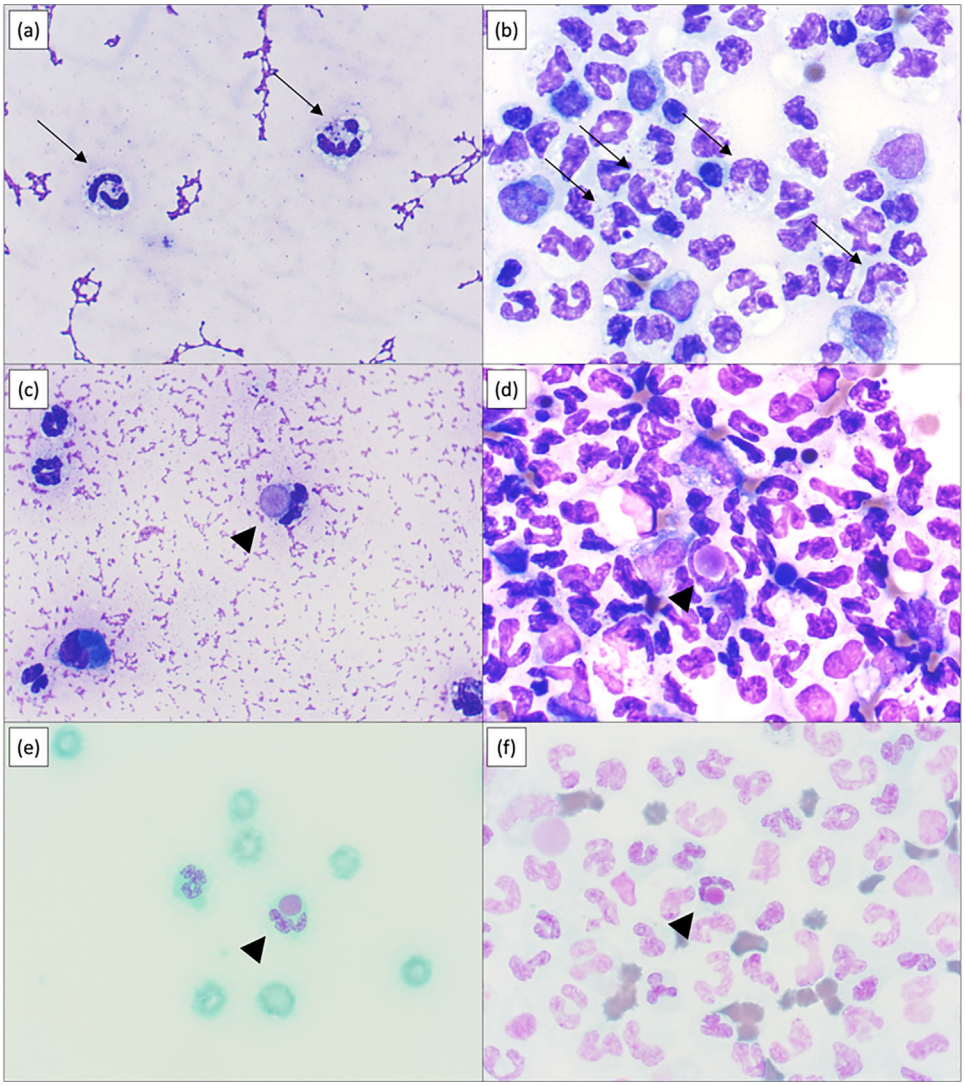

FIGURE 1 Photomicrographs of direct smears from left tarsus synovial fluid (a, c, e) and cytocentrifuged preparations from the pleural effusion (b, d, f). (a and b) Ragocytes (arrows), non-degenerate neutrophils engulfing abundant basophilic variably sized granular to globular material, May–Grünwald–Giemsa stain, 100× objective. (c and d) Lupus erythematosus (LE) cells (arrowheads), non-degenerate neutrophils containing one large round globular pale pink to purple cytoplasmic inclusion, which pushes the nucleus to the periphery, May–Grünwald–Giemsa stain, 100× objective. (e and f) The cytoplasmic inclusion within LE cells (arrowheads) demonstrates bright pink-red staining, pre-stained with May–Grünwald–Giemsa, Feulgen reaction, counterstained with 0.02% light green, 100× objective.

prednisolone (50 mg/m^2 PO SID; Prednidale, Dechra) and antimicrobial therapy (marbofloxacin 5 mg/kg PO SID; Marbocyl Vetoquinol UK, clindamycin 11 mg/kg BID PO; Zodon Ceva Animal Health), with the latter discontinued after receiving negative blood cultures.

Unfortunately, 3 weeks following discharge from the hospital, the patient re-presented due to owners' concern over poor quality of life. The patient had been seizure-free, but the owners were concerned about development of excessive panting, which was presumed to be a side effect of treatment with high doses of glucocorticoid. The owners declined the addition of a secondary immunosuppressive agent or tapering of the dose of steroid and opted for euthanasia. Postmortem examination was not performed according to the owners' wishes.

DISCUSSION

This case report describes a dog diagnosed with SLE syndrome with an atypical presentation of acute respiratory distress. SLE can present with a wide range of non-specific clinical signs, which makes diagnosis challenging, as demonstrated in this case. A thorough evaluation of the full clinical history ascertained that this dog met the revised ACR classification for SLE by fulfilling six out of the 11 criteria over the course of a lifetime (Table 1). These include the development of neurological signs, cutaneous lesions, anaemia, thrombocytopenia, serositis and a strong ANA-positive titre. However, these clinical features still overlap considerably with other infectious diseases, such as infective endocarditis, which adopts a different treatment strategy as compared to autoimmune disease. As such, it was imperative to attain more information to support the diagnosis of SLE before commencing treatment.

In this patient, the detection of LE cells in both the synovial and pleural effusion was unexpected but proved to be diagnostically significant. In dogs, LE cells are rarely identified in synovial fluid, but are highly suggestive of SLE.[13] LE cells are professional phagocytic leukocytes with a large intracytoplasmic inclusion composed of nuclear proteins, which have been opsonised by ANA or complement.[8,14] These cells are

hypothesised to form either following phagocytosis of free nuclear material, or due to autolysis of one or more lobes of the polymorphonuclear cells.[8] With routine Romanowsky stains, inclusions appear homogenous and pink to pale blue with variably defined borders and often push the nucleus to the periphery of the cell. These features allow LE cells to be distinguished from macrophages phagocytising nuclear debris (usually clumped pink to purple material). Despite the typical appearance, it is recommended to confirm the presence of DNA within the inclusions, as other phagocytised materials (e.g., certain foreign bodies/material, secretory material, etc.) may have a similar appearance. In this case, the Feulgen reaction was used for this purpose. The protocol is commonly used on fresh unstained slides; however, here it was optimised for slides pre-stained with May–Grünwald–Giemsa. As the protocol is based on a mild acid hydrolysis, the use of different dyes and fixatives may affect the results. Therefore, Feulgen reaction's protocol should be optimised for the specific laboratory conditions.

To the authors' knowledge, this is also the first confirmed case of lupus pleuritis reported in veterinary medicine. Lupus pleuritis is a common manifestation of SLE reported in up to 50% of cases in people.[15] Despite the low reported sensitivity, the detection of LE cells is considered useful in differentiating lupus pleuritis from non-SLE causes with a high specificity of 80%.[16] In this case, the development of bilateral pleural effusion resulted in the acute onset of respiratory distress, requiring emergency intervention with thoracocentesis, and the LE cells detected in the pleural effusion were indicative of lupus pleuritis. Glucocorticoid therapy was met with good response in this dog, with resolution of the clinical signs. A case report involving a 16-year-old female reported similar signs to our patient, with dyspnoea secondary to pleural effusion requiring drainage. LE cells were also detected in the pleural effusion supporting SLE diagnosis, and similarly to our case, successful treatment was achieved with glucocorticoid therapy.[17]

In SLE, the immunological hallmark is the production of ANAs,[1] and the development of ANA testing has since replaced in vitro LE cell demonstration due to the greater sensitivity and versatility. However, ANA positivity has also been noted in non-immune-mediated disorders such as those resulting in chronic inflammation (e.g., atopic dermatitis, Doberman hepatitis)[18,19] and infectious diseases (e.g., ehrlichiosis, leishmaniosis).[7] Furthermore, ANA antigens can also be detected in healthy individuals, and up to 30% of SLE-positive patients can be ANA-negative in people.[20] Assay variability has also been recognised as a major issue with our expanding knowledge of SLE, as well as the advancements in laboratory techniques. As such, positive results should be interpreted carefully alongside the full clinical presentation of the patient.

In conclusion, SLE is a complex clinical syndrome, and the diagnosis should be supported by laboratory investigations indicating immune reactivity or inflammation alongside the clinical presentation. Apart from ANA testing, early cytological evaluation of body cavity effusions can be a rapid and inexpensive option to consider, especially in atypical presentations. This will ultimately lead to prompt appropriate treatment, as demonstrated in this case. Considering the clinical history, the presence of LE cells, which was confirmed with Feulgen staining, and a strong positive ANA titre, the diagnosis of SLE in our patient was well characterised and supported.

AUTHOR CONTRIBUTIONS

Valerie S. W. Oh, Daria Starybrat, Craig R. Breheny initiated and wrote the paper, with Valerie Oh writing the main manuscript and Daria Starybrat and Craig. R Breheny involved heavily in editing. Maverick Melega and Suzanne Bussey were responsible for the cytology and laboratory interpretations and prepared the cytology images used, and also reviewed and edited the final manuscript.

ACKNOWLEDGEMENTS

We would like to acknowledge Dr. Johanna Wolf, the veterinary surgeons and nurses responsible for this dog's care.

CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

FUNDING INFORMATION

The authors received no specific funding for this work.

ETHICS STATEMENT

Full consent from the owner and the University of Edinburgh Ethics Committee has been obtained for the publication of this case report.

ORCID

Valerie S. W. Oh https://orcid.org/0009-0002-3466-8554
Craig R. Breheny https://orcid.org/0000-0002-6933-723X
Daria Starybrat https://orcid.org/0000-0001-7715-2852

REFERENCES

1. Stone M. Systemic lupus erythematosus. In: Ettinger S, Feldman E, editors. Textbook of veterinary internal medicine. 8th ed, Vol. 1. St. Louis, MO: Saunders Elsevier; 2017: p. 873–77.
2. Berent A, Cerundolo R. Systemic lupus erythematosus. Compend standards of care. Emerg Crit Care Med. 2005;7:7–11.
3. Camus MS, Almy FS, Cienava EA. A ragtime joint. Immune-mediated polyarthropathy in a dog. Vet Clin Pathol. 2010;39(1):4.
4. Kilkenny E, Watson C, Dukes-McEwan J, Bode EF, Hezzell MJ, Payne JR, et al. Evaluation of serum cardiac troponin-I concentrations for diagnosis of infective endocarditis in dogs. J Vet Intern Med. 2021;35(5):2094–101.
5. Meléndez-Lazo A, Fernández M, Solano-Gallego L, Pastor J. What is your diagnosis? Synovial fluid from a dog. Vet Clin Pathol. 2015;44(2):329–30.
6. Tan EM, Cohen AS, Fries JF, Masi AT, McShane DJ, Rothfield NF, et al. The 1982 revised criteria for the classification of systemic lupus erythematosus. Arthritis Rheum. 1982;25(11):1271–77.
7. Smith BE, Tompkins MB, Breitschwerdt EB. Antinuclear antibodies can be detected in dog sera reactive to *Bartonella vinsonii* Subsp. *berkhoffii*, *Ehrlichia Canis, or Leishmania Infantum* antigens. J Vet Intern Med. 2004;18(1):47–51.
8. Hepburn AL. The LE cell. Rheumatology. 2001;40(7):826–27.
9. Park JY, Malik A, Dumoff KL, Gupta PK. Case report and review of lupus erythematosus cells in cytology fluids. Diagn Cytopathol. 2007;35(12):806–9.
10. Nosanchuk JS, Kim CW. Lupus erythematosus cells in CSF. JAMA. 1976;236(25):2883–84.
11. Schumacher HR Jr, Howe HS. Synovial fluid cells in systemic lupus erythematosus: light and electron microscopic studies. Lupus. 1995;4(5):353–64.
12. Black LJ, Hechler AC, Duffy ME, Beatty SSK. Presumed lupus erythematosus cells identified in bronchoalveolar lavage fluid from a Mexican hairless dog. Vet Clin Pathol. 2017;46(2):354–59.
13. Pederson NC. A review of immunologic diseases of the dog. Vet Immunol Immunopathol. 1999;69(2):251–342.
14. Hargraves MM. Discovery of the LE cell and its morphology. Mayo Clin Proc. 1969;44(9):579–99.

15. Orens, JB, Martinez, FJ, Lynch JP. Pleuropulmonary manifestations of systemic lupus erythematosus. Rheum Dis Clin North Am. 1994;20(1):159.
16. Choi BY, Yoon MJ, Shin K, Lee YJ, Song YW. Characteristics of pleural effusions in systemic lupus erythematosus: differential diagnosis of lupus pleuritis. Lupus. 2015;24(3):321–26.
17. Gulhane S, Gangane N. Detection of lupus erythematosus cells in pleural effusion: an unusual presentation of systemic lupus erythematosus. J Cytol. 2012;29(1):77–79.
18. Dyggve H, Meri S, Spillmann T, Jarva H, Speeti M. Antihistone autoantibodies in dobermans with hepatitis. J Vet Intern Med. 2017;31(6):1717–23.
19. Ginel PJ, Lucena R. Investigation of antinuclear antibodies in canine atopic dermatitis. J Vet Med A Physiol Pathol Clin Med. 2001;48(4):193–98.
20. Pisetsky DS, Rovin BH, Lipsky PE. New perspectives in rheumatology: biomarkers as entry criteria for clinical trials of new therapies for systemic lupus erythematosus: the example of antinuclear antibodies and anti-DNA. Arthritis Rheumatol. 2017;69(3):487–93.

How to cite this article: Oh VSW, Melega M, Bussey S, Breheny CR, Starybrat D. Diagnosis of systemic lupus erythematosus in a dog with an atypical presentation of pleuritis. Vet Rec Case Rep. 2023;e747. https://doi.org/10.1002/vrc2.747