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CASE REPORT

Presumed pseudo-Pelger–Huët anomaly and basophilia secondary to chronic lymphocytic leukemia in a dog

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Abstract

A 10-year-old neutered male Maltese dog was presented for an investigation of lymphocytosis. The dog was up-to-date on vaccinations and deworming. Physical examination did not reveal any significant abnormalities. A complete blood cell count (CBC) showed mild leukocytosis with moderate lymphocytosis, basophilia, and moderate neutropenia, but no significant left shift or toxic change. Serum biochemistry and urinalysis were unremarkable. All performed tests for infectious agents common in this geographical region were negative. No significant abnormalities were found on abdominal ultrasound examination. Multiparametric flow cytometry of peripheral blood showed a $CD8^+$ T-cell lymphocytosis, and PCR for antigen receptor rearrangement revealed a clonal expansion of the T-cell receptor gamma chain genes. A clinical diagnosis of chronic lymphocytic leukemia (CLL) was made, and follow-up was recommended. On Day 48 post-presentation, the CBC showed mild non-regenerative anemia (NRA), moderate leucocytosis due to moderate to marked lymphocytosis, basophilia, and a marked increase in hyposegmented neutrophils with mild toxic change in the absence of neutrophilia or neutropenia. Treatment with chlorambucil and prednisolone was initiated. On Days 87 and 197 post-presentation, the CBC showed mild NRA, with progressively decreasing numbers of hyposegmented neutrophils. The dog remained without clinical signs. Basophilia and probable pseudo-Pelger–Huët anomaly were possibly secondary to CLL. To the authors' knowledge, this is the first report of these two hematologic conditions secondary to CLL in dogs. Recognition of a pseudo-Pelger–Huët anomaly is clinically relevant to avoid misinterpretation as a marked left shift due to severe inflammation and prevent unnecessary urgent therapeutic actions.

KEYWORDS
basophils, canine, flow cytometry, hematology, hyposegmented granulocytes, lymphoproliferative disease

1 | CASE PRESENTATION

A 10-year-old neutered male, 5.7 kg weight, Maltese dog, was referred to the internal medicine service (Day 0) for evaluation of lymphocytosis, which was detected by the referring veterinarian during a routine blood test. The dog was up-to-date on vaccinations and deworming by the referring veterinarian. Physical examination revealed no significant abnormalities.

A complete blood cell count (CBC) was performed (Table 1; Figure 1) using Sysmex XN-1000V (Sysmex Corporation, Norderstedt, Germany) following peripheral blood (PB) smear examination with manual differential white blood cell count. The CBC revealed mild leukocytosis (19.2, reference interval [RI]: 6–17 $\times 10^9$ cells/L) with moderate lymphocytosis (14.6, RI: 1–4.8 $\times 10^9$ cells/L), basophilia (2.1, RI: 0–0.2 $\times 10^9$ cells/L), and neutropenia (1.3, RI: 3–11.5 $\times 10^9$ cells/L) without significant left shift or toxic change. Lymphocytes were small to intermediate in size, with scant to slightly increased amounts of pale blue cytoplasm, and frequently, they had cytoplasmic pinkish to magenta granules. Some particle misclassifications by the automated analyzer were detected on the WDF and RET-EXT channels, which are discussed further below (Figure 1). Serum biochemistry, including basal cortisol concentration (Table S1), was unremarkable. Urinalysis was performed on a cystocentesis-obtained sample and revealed pale yellow urine with mild turbidity, pH 5, specific gravity >1.050, trace urine protein, and negative results for other analytes. The urine sediment examination was unremarkable. Abdominal ultrasound did not reveal any significant abnormalities.

All performed tests for infectious agents common in this geographical region were negative: serology for *Leishmania* spp. (ELISA); for *Ehrlichia canis* and *Ehrlichia ewingii*, *Borrelia burgdorferi*, *Anaplasma phagocytophilum*, and *Anaplasma platys* and antigen detection of *Dirofilaria immitis* (SNAP 4Dx Plus Test, IDEXX Laboratories); and PCR for *Babesia* spp. and *Theileria* spp.

Peripheral blood was analyzed by multiparametric flow cytometry (Figure S1) on a CytoFLEX LX Flow Cytometer (Beckman Coulter Inc., Brea, CA, USA) using conventional protocols previously described[1] and using canine-specific and cross-reactive monoclonal antibodies against canine cluster of differentiation (CD)45, CD18, CD21, CD5, CD3, CD4, CD8, CD25, CD14, CD34, major histocompatibility complex class II (MHCII), and Ki67 (Table S2). CytExpert Software (Beckman Coulter Inc., Brea, CA, USA) was used to interpret the flow cytometry data. Different leukocyte populations were established based on light scatter and immunophenotype properties (backgating) of viable single events using the viability dye 7-aminoactinomycin D (7AAD): "lymphocytes" and "granulocytes and monocytes." Cells classified as lymphocytes (68%) were positive for CD45 [93%, of which were positive for CD5 (96%, of which were positive for CD8 (89%))], CD18 (82%), CD3 (90%), and MHCII (93%); and were negative for CD21, CD34, CD25, CD14, and Ki67. These results were interpreted as a $CD8^+$ T-cell lymphocytosis. PCR for antigen receptor rearrangement (PARR) test was performed on EDTA PB at IDEXX Laboratories (Barcelona, Spain) and revealed a clonal expansion of T-cell receptor gamma chain genes and polyclonal immunoglobulin heavy-chain gene rearrangement.

TABLE 1 Complete blood cell count (CBC) performed on the Sysmex XN-100V with peripheral blood (PB) smear review.

Parameter (units)	Reference interval	Presentation (Day 0)	Day 48 post-presentation	Day 87 post-presentation	Day 197 post-presentation
PCV (L/L)	0.37–0.55	0.38	**0.34**	**0.32**	**0.35**
Red blood cell (RBC) count (10^{12} cells/L)	5.5–8.5	5.28	**4.74**	**4.49**	**5.26**
Hemoglobin (g/L)	120–180	126	**115**	**110**	126
Reticulocyte count (10^9 cells/L)	0–60	36.43	52.61	35.02	42.08
White blood cell (WBC) count (10^9 cells/L)	6–17	**19.24**	**36.7**	8.28	8.54
Segmented neutrophils (10^9 cells/L)	3–11.5	**1.35**	7.52	4.02	4.78
Hyposegmented neutrophils (10^9 cells/L)	0–0.3	0	**4.04**	**0.5**	0.21
Lymphocytes (10^9 cells/L)	1–4.8	**14.62**	**21.47**	2.32	2.05
Monocytes (10^9 cells/L)	0.15–1.35	0.58	1.1	1.04	1.32
Eosinophils (10^9 cells/L)	0.1–1.5	0.58	1.1	0.41	0.17
Basophils (10^9 cells/L)	0–0.2	**2.12**	**1.47**	0	0
Platelets (10^9 cells/L)	200–500	315	447	478	402

Note: The differential white blood cell (WBC) count was calculated using the percentages from the PB smear and the total WBC count from the CBC. Altered parameters in bold. Comments on PB smear review: Day 0, small to medium-sized lymphocytes, often granulated; Day 48, marked hyposegmentation of neutrophils (with mild toxic change) and eosinophils, similar cytomorphology of lymphocytes to that of Day 0, rouleaux formation of RBC; Day 87: slightly increased hyposegmented neutrophils with mild toxic change; Day 197: rouleaux formation of RBC.

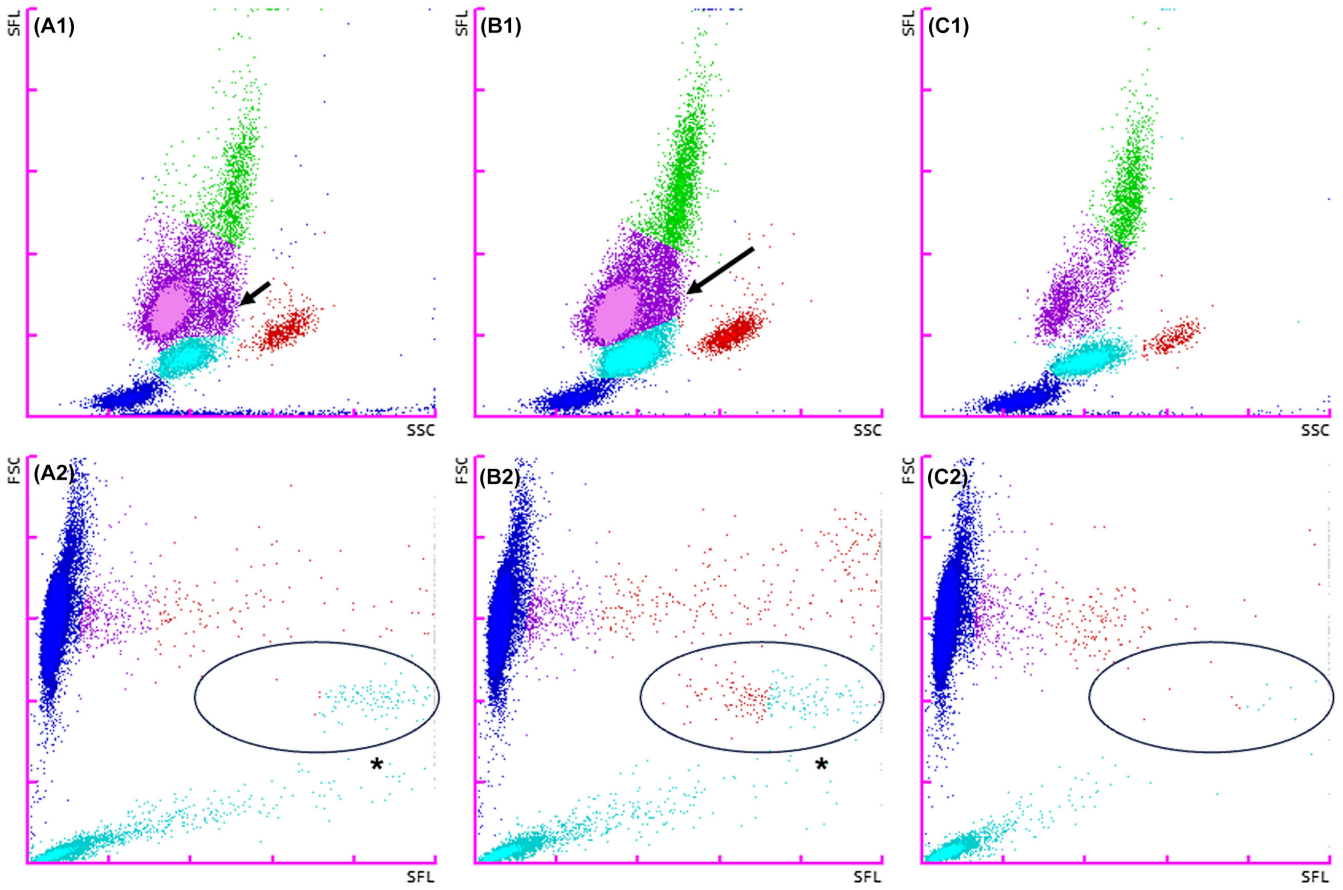

FIGURE 1 Scattergrams of WDF (A1, B1, C1) and RET-EXT (A2, B2, C2) channels from the Sysmex XN-1000V analyzer of peripheral blood from a dog with pseudo-Pelger–Huët anomaly on Day 0/initial presentation (A1, A2), Day 48 post-presentation (B1, B2) and Day 197 post-presentation (C1, C2). Particle representation in WDF: dark blue dots (debris), clear blue dots (neutrophils), purple dots (lymphocytes), green dots (monocytes), and red dots (eosinophils). Particle representation in RET-EXT: dark blue dots (mature red blood cells), purple dots (low-fluorescence reticulocytes), red dots (immature [medium- and high-] fluorescence reticulocytes), and clear blue dots (platelets). The short arrow probably represents the basophils identified on the peripheral blood smear examination. A similar area indicated by the long arrow is thought to represent both basophils and hyposegmented neutrophils. Ellipses marked with an asterisk could represent either small cytoplasmic fragments of neoplastic cells or neoplastic cells themselves (less likely).

A clinical diagnosis of chronic lymphocytic leukemia (CLL) was made, and monthly follow-up was recommended. The moderate neutropenia was not further investigated at that time because the dog had no clinical signs, no left shift, and no toxic changes in neutrophils.

On Day 48, after the initial presentation, the dog remained asymptomatic. The CBC was repeated (Table 1; Figure 1) and showed mild non-regenerative anemia (NRA) (Pack cell volume [PCV] 0.34, RI: 0.37–0.55 L/L), moderate leukocytosis (36.7, RI: 6–17 $\times 10^9$ cells/L) due to moderate to marked lymphocytosis (21.5, RI: 1–4.8 $\times 10^9$ cells/L), moderate basophilia (1.5, RI: 0–0.2 $\times 10^9$ cells/L), and increased hyposegmented neutrophils (4, RI: 0–0.3 $\times 10^9$ cells/L) in the absence of neutrophilia or neutropenia (Figure 2). Lymphocytes were similar to those described on Day 0, with frequent cytoplasmic granules. Hyposegmentation was evident in both neutrophils (with no to minimal toxic change) and eosinophils. The hyposegmentation was limited to nuclei with band-shaped morphology, without the presence of round-shaped nuclei. The chromatin pattern appeared clumped, coarse, and mature. At this point, the described hematologic findings were interpreted as probable pseudo-Pelger–Huët anomaly and basophilia, possibly secondary to the CLL of the dog.

Treatment was started with oral chlorambucil at a dose of 6 mg/m^2 body surface area once daily for 10 days, then changed to every other day. Oral prednisolone was started at 1 mg/kg body weight once daily for 10 days, then decreased to 0.7 mg/kg once daily for 10 days and later decreased to 0.7 mg/kg every other day. A medical review was scheduled for 1 month later.

At follow-up visits on Days 87 and 197 after initial presentation the owners reported that their dog remained without clinical signs with a mild increase in appetite. The CBC was repeated (Table 1) and showed mild NRA (PCV 0.32, RI: 0.37–0.55 L/L) and low numbers of hyposegmented neutrophils (0.5, RI: 0–0.3 $\times 10^9$ cells/L) in absence of significant toxic change on Day 87, and mild NRA (PCV 0.35, RI: 0.37–0.55 L/L) on Day 197 (Figure 1). Basophilia and lymphocytosis were absent on both days. It was recommended to continue the treatment as it was well tolerated, and the response was good. Active

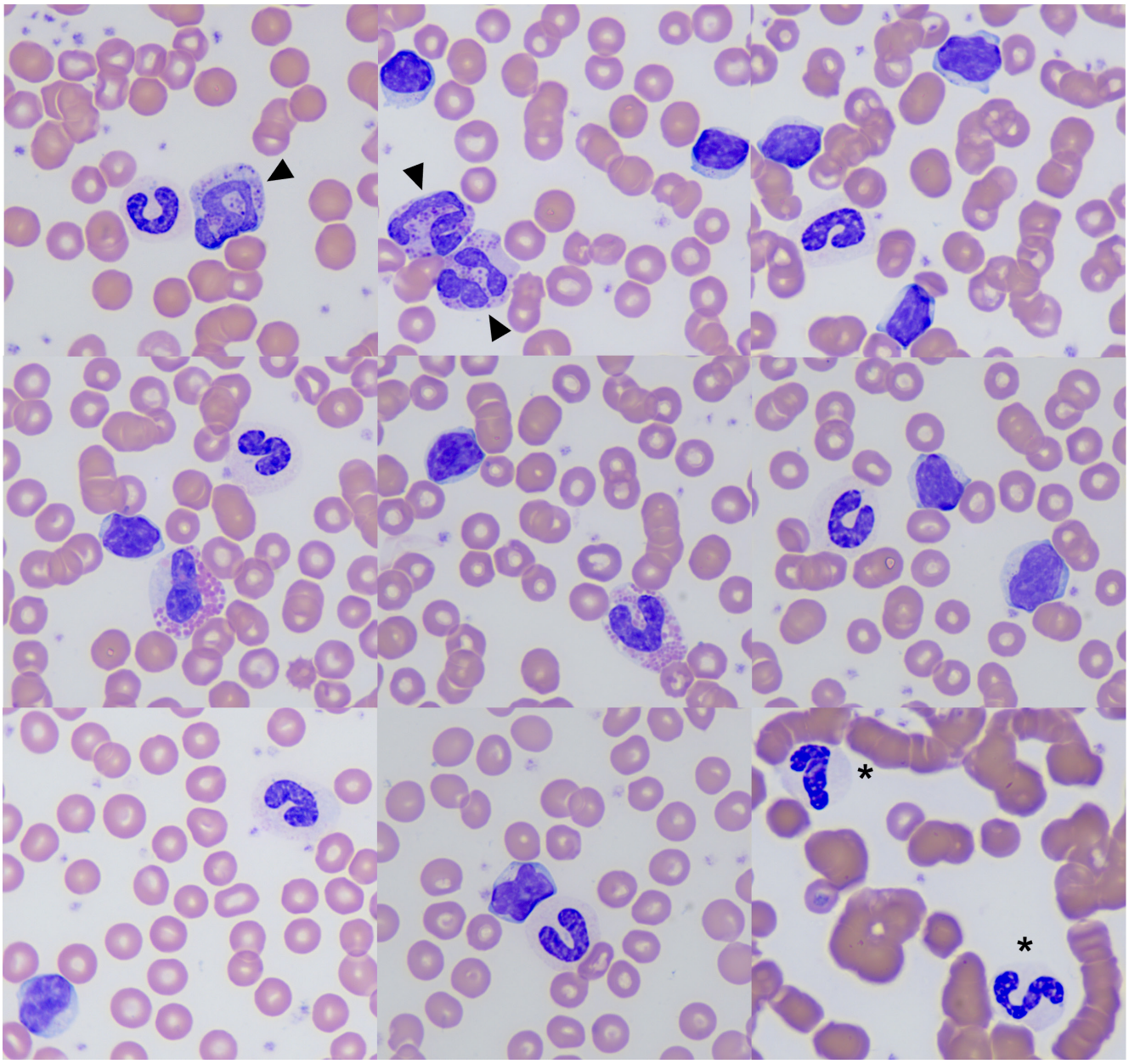

FIGURE 2 Peripheral blood smear micrographs on Day 48 from a dog with pseudo-Pelger–Huët anomaly, showing hyposegmented neutrophils and eosinophils, basophils (arrowhead), lymphocytes and segmented neutrophils (asterisks). Modified Wright stain, original magnification ×100 objective.

surveillance (physical examination and CBC) every 3–6 months was recommended.

2 | DISCUSSION

Pelger–Huët anomaly has been described in humans, dogs, cats, rabbits, and horses. Neutrophils (and other cells such as eosinophils, basophils, or megakaryocytes) appear with a hypolobulated or even round nuclei due to a failure in nuclear segmentation, with a clumped chromatin pattern and absence of toxic change.[2,3] In humans, the underlying causative mutation involves the gene encoding the Lamin B receptor (an integral membrane protein in the nuclear envelope). However, the genotype associated with the Pelger–Huët phenotype in animals was not known.[2] While in humans, this condition has an autosomal dominant inheritance pattern, in animals, this inherited disorder has traditionally been considered an autosomal dominant trait with incomplete penetrance, in which heterozygous animals have the typical phenotype (hyposegmentation) but are usually without clinical signs.[2,3] In Australian Shepherd dogs, Pelger–Huët anomaly is now known to be caused by a splice site mutation in the LMBR1L (Limb Development Membrane Protein 1 Like) gene and has an autosomal recessive mode of inheritance.[4]

In contrast to the typical Pelger–Huët anomaly, an acquired condition (pseudo-Pelger–Huët anomaly) has been described that is usually transient and secondary to inflammation, myeloid neoplasms, administration of certain drugs or myelodysplasia with asynchronous neutrophil maturation.[2,3] Typically, cases of pseudo-Pelger–Huët anomaly show only a minority of hyposegmented cells, as opposed to the inherited form in which most cells are affected.[2] Clinically, it is important to differentiate between inherited Pelger–Huët anomaly (non-pathological) and pseudo-Pelger–Huët anomaly, which is secondary to pathological processes and requires additional diagnostic investigation. It may also be relevant to recognize Pelger–Huët anomaly and acquired Pelger–Huët anomaly from other conditions, such as a marked or degenerative left shift due to severe inflammation. In such cases, significant toxic change would typically be expected in addition to the hyposegmented neutrophils, and urgent diagnostic investigation is required. In this case, the presence of an acquired Pelger–Huët anomaly is supported by the absence of hyposegmented neutrophils at initial presentation, the characteristic clumped mature chromatin of the hyposegmented granulocytes, and the variable number of these cells in the subsequent CBC.

There are some descriptions of pseudo-Pelger–Huët anomaly in cases of human CLL.[5,6] To the authors' knowledge, this case is the first description of this association in the canine species. It is not entirely clear whether this acquired condition is due to impaired myelopoiesis because of bone marrow infiltration with the neoplastic lymphocytes[6] or due to chemotherapy.[5] In our case, it seems that chemotherapy did not contribute to the formation of hyposegmented leukocytes since this finding was present before the start of treatment. In addition, the number of hypolobulated neutrophils decreased significantly on Day 87 post-presentation and finally resolved on Day 197 post-presentation. The authors hypothesize that nontreatment-related causes may have contributed to the hyposegmentation of leukocytes, and the possible different considerations were bone marrow infiltration with neoplastic lymphocytes (although bone marrow cytology was not performed), impaired myelopoiesis due to an excessive peripheral neutrophil demand (it should be considered because neutropenia was present at the initial presentation) or myelodysplasia with asynchronous neutrophil maturation for some other unknown reason. However, the fact that hyposegmented neutrophils were not seen in the presence of neutropenia at initial presentation is of unknown significance, and a transient decreased granulopoiesis or an acute/hyperacute demand/consumption of neutrophils could be some possible explanations.

Basophils, like mast cells, are involved in T-helper lymphocyte Type 2 mediated immunity and in Type 1 hypersensitivity reactions. Interleukin 3 is the major basophil growth factor, promoting basophil production, differentiation, and survival.[7] In dogs, basophilia is usually accompanied by eosinophilia, and both share similar etiologies, including endo/ectoparasites, hypersensitivity,[8] eosinophilic lung diseases (eosinophilic bronchitis, granuloma, and/or broncho-pneumopathy),[9] fungal/algal disease, paraneoplastic disease, or primary neoplasia (eosinophilic or basophilic leukemia).[8] Paraneoplastic basophilia has also been described in thymoma,[10,11] mast cell neoplasia,[12,13] T-cell lymphoma,[14] lymphomatoid granulomatosis,[15] and in several myeloproliferative neoplasms, including essential thrombocythemia,[16,17] idiopathic/primary canine myelofibrosis,[18] myeloid leukemia, and polycythemia vera.[7] This case seems to be the first reported paraneoplastic peripheral basophilia secondary to CLL in a dog. It is partially supported by the fact that paraneoplastic conditions, by definition, resolve when the causative disease is in remission,[19] as appears to be the situation on Days 87 and 197 post-presentation. However, in this case, the status of minimal residual disease was not further investigated, and flow cytometry or PARR of PB or bone marrow after initiation of treatment could have helped resolve this dilemma. Nevertheless, dogs with CLL rarely achieve a true, complete remission, but rather a partial remission with an attempt at palliative treatment and always have a final fatal course.[20]

CLL is a lymphoproliferative disease of mature lymphocytes that primarily affects adult dogs (7–13 years of age) and is usually indolent, with reported median survival times of 930–1098 days. The T-cell phenotype is the most common, accounting for 60%–90% of all CLL. Flow cytometry of T-cell CLL is characterized by CD3 and/or CD5-positive cells, usually expressing CD8 (consistent with the granular cytologic morphology) and MHCII, although CD4 and CD8 positivity may be variable.[21]

Lymphocyte counts in T-cell CLL are typically $10–50\times10^9$ cells/L but can sometimes be as high as $>500\times10^9$ cells/L.[21] In this case, given the moderate degree of lymphocytosis, the diagnosis of CLL was made after the demonstration of a homogeneous lymphocyte phenotype ($CD8^+$ T cells), clonal expansion of T-cell receptor gamma chain genes by PARR, and exclusion of hypoadrenocorticism, common pathogens, inflammatory conditions, or evident tissue involvement. The absence of an overt organomegaly or lymph node enlargement makes lymphoma with hemic extension less likely. Fine-needle aspiration of the spleen was not performed due to the lack of splenomegaly. However, T-zone lymphoma with blood involvement could be another differential diagnosis in a dog with clonal T-cell lymphocytosis, which was ruled out in our case due to the phenotypic characteristics (absence of a substantial CD45-negative T-cell population). In dogs, some infectious diseases have been associated with $CD8^+$ T-cell lymphocytosis, particularly *E. canis*, resulting in lymphocyte counts up to 17×10^9 cells/L, sporadically up to 30×10^9 cells/L.[22] No antibodies against *E. canis* were found in this dog. Other causes of lymphocytosis include hypoadrenocorticism, which was ruled out as the basal serum cortisol concentration was >55 nmol/L (>2 μg/dL); or thymoma, which is expected to have a mixed or double-negative $CD4^-/CD8^-$ T-cell lymphocytosis.[22,23]

When CLL is found incidentally, and no significant clinical signs or severe hematologic abnormalities are identified, the initial recommendation is active surveillance of the dog rather than active treatment.[20] Besides the clinical deterioration of the dog, the presence of an abnormal hematologic result is one of the reasons used at our institution to initiate treatment of CLL. In this case, treatment was

initiated on Day 48 after initial presentation due to the hematologic changes (presence of anemia, significant increase in lymphocyte count, persistence of basophilia, and granulocyte hyposegmentation) together with the owner's choice of a more interventionist approach. Some particle misclassifications by the Sysmex XN-1000V automated analyzer were detected on WDF and RET-EXT channels (Figure 1). On the WDF scattergram, the short arrow (Figure 1A1) depicts a purple, distinct, smaller population right to the usual lymphocyte population on the left and probably represents the basophils[24–26] found on the PB smear evaluation (11%). As previously reported,[24,26] basophils were also not detected on the WNR channel (data not shown). The long arrow (Figure 1B1) shows an almost indistinct population similar to that described in Figure A1, which could represent mostly basophils and hyposegmented neutrophils, considering the reported data[25,26] and the results of PB smear. The ellipse on the RET-EX scattergrams (Figure 1A2–C2) delineates a distinct particle population identified by the analyzer as platelets and immature-reticulocytes. This was particularly evident at initial presentation and Day 48 post-presentation (Figure 1, asterisks) when the dog had lymphocytosis. However, given the diagnosis of CLL in this dog, these particles may be either cytoplasmic fragments of neoplastic cells or neoplastic cells themselves.[26–28] In the authors' opinion, the latter seems less likely in this case as the size (*y*-axis of the RET-EX scattergram [forward scatter]) of the encircled particle population is similar to that of the smallest red blood cells. Nevertheless, this particle misidentification was not clinically significant and did not impact diagnosis or any clinical decisions since they were present in relatively low numbers and did not affect the reticulocyte or platelet counts beyond their reference intervals.

We describe two hematologic alterations (basophilia and pseudo-Pelger–Huët anomaly) that are probably associated with CLL in a dog. However, further studies are needed to fully understand the mechanisms leading to increased basophil concentration and granulocyte hyposegmentation in canine CLL. Recognition of pseudo-Pelger–Huët anomaly is clinically relevant. Misinterpretation as a marked and/or degenerative left shift due to severe inflammation could lead to unnecessary urgent therapeutic actions.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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SUPPORTING INFORMATION

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

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