CASE REPORT

A case of leukaemia cutis in a dog with T-cell chronic lymphocytic leukaemia

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Leukaemia cutis (LC) is the infiltration of neoplastic leukocytes into the skin, characterised by haemorrhagic papules, nodules, and plaques. LC has been reported in human leukaemia patients, but it is extremely rare in dogs. A 13-year-old spayed female Golden Retriever that was previously diagnosed with chronic lymphocytic leukaemia was managed with chlorambucil (20 mg/m² orally, every 2 weeks) and prednisolone (2 mg/kg orally, every other day) for 8 months; however, immunosuppression was temporarily discontinued because of a bacterial urinary tract infection. Cutaneous signs, including multifocal ecchymosis and white plaques, appeared 1 month after cessation of chemotherapy. Histopathological examination revealed small- to intermediate-sized lymphocytes with mild atypia in a perivascular to interstitial pattern within the superficial dermis. The bands of atypical cells within the superficial dermis were strongly and extensively positive for CD3 on immunohistochemistry. Polymerase chain reaction analysis of the biopsied skin revealed clonal rearrangement of the T-cell receptor gamma locus gene. Given the evidence of clinical signs, peripheral immunophenotyping, histopathology, immunohistochemistry, and clonal gene arrangement, LC was diagnosed. The lesions disappeared when chemotherapy was restarted but were occasionally observed when chemotherapy was stopped. To the authors’ best knowledge, this is the first case report of LC in a dog.

KEYWORDS  
chronic lymphocytic leukaemia, cutaneous manifestation, immunohistochemistry, leukaemia cutis, T-cell

1 | INTRODUCTION

Chronic lymphocytic leukaemia (CLL), a haematological disorder affecting middle-aged and geriatric dogs, is characterised by abnormal neoplastic clonal proliferation of morphologically small, mature lymphocytes with persistent lymphocytosis (Workman & Vernau, 2003). In dogs, CLL is frequently asymptomatic; however, it can induce peripheral lymphadenopathy, hepatosplenomegaly, lymphocytosis, anaemia, thrombocytopenia, and increased lymphocyte proliferation in the bone marrow. In contrast with human CLL cases, 95% of which involve B-cell lineages, T-cell CLL is far more common than B-cell CLL in dogs (Vernau & Moore, 1999; Workman & Vernau, 2003).

Cutaneous manifestations are seen in human leukaemia and involve non-specific skin lesions, such as paraneoplastic vasculitis, infections,
insect bite/insect bite-like reactions, and paraneoplastic pemphigus (Martínez-Leboráns et al., 2016; Robak & Robak, 2007), or less commonly, direct cutaneous infiltration of leukaemic cells (leukaemia cutis, LC) (Ali et al., 2006). LC lesions involve haemorrhagic papules, ecchymosis, nodules, and plaques (Beswick et al., 2002; Cho-Vega et al., 2008; Robak & Robak, 2007; Paydaş & Zorludemir, 2000). LC occurs more often in human patients with acute myeloid leukaemia (Martínez-Leboráns et al., 2016) and uncommonly in patients with chronic myeloproliferative disorders, including lymphocytic leukaemia (Cho-Vega et al., 2008). In human lymphocytic leukaemia, skin involvement is observed in T-cell lymphoblastic leukaemias/lymphomas, rather than in B-lymphocyte CLL (Ali et al., 2006; Sander et al., 1991; Yen et al., 1996).

To the best of our knowledge, LC accompanying CLL has never been reported in veterinary medicine. Herein, we report a canine case of CLL presenting with ecchymosis and multifocal white plaques in the skin, which was defined as LC.

2 | CASE DESCRIPTION

A 13-year-old spayed female Golden Retriever was presented to the veterinary medical teaching hospital for persistent peripheral lymphocytosis. The dog exhibited mild generalised lymphadenopathy but was otherwise asymptomatic. A complete blood count revealed leucocytosis (67.2 × 10⁹/L; reference range, 5.05–16.76 × 10⁹/L) with lymphocytosis (58.32 × 10⁹/L; reference range, 1.05–5.1 × 10⁹/L). Microscopy of the peripheral blood showed predominantly small- to intermediate-sized lymphocytes with normal morphology. Immunophenotyping showed CD3⁺/CD5⁺/CD4⁻/CD8⁻/CD21⁻/CD79a⁻/CD34⁻ cells (Figure 1). Accordingly, the patient was diagnosed with T-cell CLL. Peripheral lymphocytosis (>60 × 10⁹/L) had persisted for more than 3 months (range, 58.32–81.53 × 10⁹/L), and mild non-regenerative anaemia (packed cell volume, 31.8%; reference range, 37.3–61.7%) was present at 3 months after the first visit to the veterinary medical teaching hospital.

Chlorambucil (20 mg/m² orally, every 2 weeks) and prednisolone (2 mg/kg orally, every other day) were administered, and the CLL was well-managed for 8 months until a multidrug-resistant Klebsiella pneumoniae infection was isolated in the urinary tract. Based on the culture and sensitivity test, the following antibiotics were used: cefixime, enrofloxacin, marbofloxacin, cefovecin, amikacin, amoxicillin-clavulanate, vancomycin, and imipenem. After the chemotherapy had been discontinued for 1 month due to the Klebsiella infection, new dermatological lesions of multifocal purpura and ecchymoses developed bilaterally in the inguinal areas, thighs, and around the neck. Thereafter, multiple white plaques were noted on the right side of the neck (Figure 2). No mucus membrane lesions were found.

No abnormal findings were observed in platelet counts (387 × 10⁹/L; reference range, 148–484 × 10⁹/L), buccal membrane bleeding time (less than 3 min), prothrombin time (12 s; reference range, 11–17 s), or activated partial thromboplastic time (80 s; reference range, 71–102 s), ruling out haemostatic disorders. Thromboelastography showed a normal coagulation pattern. Comprehensive serum biochemistry analysis was not remarkable: alanine aminotransferase (10 U/L, reference range 10–125 U/L), alkaline phosphatase (62 U/L, reference range 23–212 U/L), urea nitrogen (6.07 mmol/L, reference range 2.5–9.6 mmol/L), creatinine (79.6 µmol/L, reference range 44.2–159.3 µmol/L), glucose (5.6 mmol/L, reference range 3.9–7.9 mmol/L), total protein (71 g/L, reference range 52–82 g/L), and globulin (41 g/L, reference range 25–45 g/L).

Dermatological cytology revealed no infectious agents. Biopsy specimens were obtained from the white plaques on the neck and the areas of purpura and ecchymosis in the inguinal area. Microscopy revealed lichenoid infiltration of a uniform population of small- to intermediate-sized lymphocytes in the superficial dermis with a perivascular to interstitial pattern (Figure 3a). The infiltrated lymphocytes showed mild nuclear and cytoplasmic pleomorphism, with rare mitotic figures.
FIGURE 2  (a–d) Multifocal purpura and ecchymoses are observed in the inguinal areas. (e and f) Multiple white plaques are noted on the right side of the neck

Epitheliotropism was not observed. Multifocal haemorrhage and oedema were present in the superficial and periadnexal dermis.

Immunohistochemistry was performed using an anti-CD3 rabbit polyclonal antibody (DAKO, Santa Clara, CA, USA) and an anti-CD79a mouse monoclonal antibody (Bio-Rad, Hercules, CA, USA). The atypical cell bands in the superficial dermis were strongly and extensively positive for CD3, but negative for CD79a, confirming their T-cell origin (Figure 3b). Polymerase chain reaction (PCR) for antigen receptor rearrangement testing from formalin-fixed tissue (VDx Veterinary Diagnostics and Preclinical Research Services, Davis, CA, USA) revealed the clonal rearrangement of the T-cell receptor gamma locus.

Based on clinical signs, peripheral immunophenotyping, histopathology, immunohistochemistry, and clonality tests, we concluded that the intradermal monomorphic cellular infiltrate may be a cutaneous manifestation of lymphocytic leukaemia, or LC. The dog was retreated with chemotherapeutic agents, chlorambucil (20 mg/m² orally, every 2 weeks), and prednisolone (2 mg/kg orally, every other day) for a month, and the skin lesions disappeared. During follow-up, chemotherapy was occasionally administered over 9 months because of the persistent urinary tract infection. At 17 months after diagnosis, the dog had worsened non-regenerative severe anaemia due to chronic kidney disease, and the owner decided not to continue aggressive treatment. The skin lesions disappeared whenever chlorambucil and prednisolone were administered but recurred when both drugs were stopped.

3  DISCUSSION

Dermatological signs of leukaemia are unusual and mostly involve non-specific changes. LC is a specific leukaemia-associated dermatological change and has not previously been reported in dogs to the best of our knowledge. Since leukaemic cells can be identified in unspecific skin changes seen in leukaemia and other skin diseases such as herpes simplex lesions, psoriasis vulgaris, and various epidermal neoplasms in human (Dargent et al., 1998; Metzler et al., 1997; Smoller & Warnke, 1998; Wagner et al., 2012; Ziemer et al., 2005), LC cannot be diagnosed solely based on skin lesions; instead, laboratory tests, including histopathology, immunohistochemistry, and lymphocyte clonality tests are required. Even in humans, there is no specific clinical morphological appearance based on the type of leukaemia and infiltrated neoplastic cells (Wagner et al., 2012). Because of the lack of information on LC in dogs with leukaemia, we diagnosed LC by excluding other differential diagnoses and based on the histopathology and immunohistochemistry results.

The patient initially presented with ecchymosis after the development of white plaques. Plaques may appear with chronic inflammatory disease. The differential diagnoses of ecchymosis in dogs are usually coagulopathies, such as platelet disorders, vascular disorders, coagulation disorders, and trauma (Miller et al., 2012). Trauma and primary haemostatic disorders were excluded through patient history and buccal mucosal bleeding time. Coagulation tests were all confirmed to be normal. Drug-induced hypersensitivity is also one of the differential diagnoses for petechiae and ecchymoses, although it is rarely seen in dogs (Zachary, 2017). However, histopathologic findings are unlike those in this case, and the skin lesions were not affected by antibiotic administration but mainly by chlorambucil. Type IV hypersensitivity, a T-lymphocyte-mediated response, could have similar skin lesions. However, intensively infiltrating homogeneous lymphocytes derived from T-cells, and the lack of inflammatory cells, such as neutrophils, mononuclear cells, or eosinophils, in the perivascular lesion support the finding that the lesion is less likely to be type IV hypersensitivity. Ultimately, the skin biopsy revealed LC lesions of lichenoid infiltration of small- to intermediate-sized neoplastic lymphocytes in a perivascular to interstitial pattern. Lymphocytes strongly expressed CD3 and the T-cell receptor gamma locus on PCR, indicating that dermal infiltrates were likely CLL-associated neoplastic T-lymphocytes.
In humans, LC of T-cell origin leukaemias/lymphomas occurs in precursor T-cell acute lymphocytic leukaemia (pre-T-ALL), adult T-cell leukaemia/lymphoma (ATLL), T-cell prolymphocytic leukaemia (T-PLL), and Sézary syndrome (SS) (Cho-Vega et al., 2008). Since there is little information regarding LC in dogs, it was difficult to clearly apply the classification criteria of humans to this case; however, when the classification based on the clinical signs, including cutaneous involvement and phenotype, was applied, T-PLL was considered to be the most similar. It is characterised by peripheral lymphocytosis, hepatosplenomegaly, and generalised lymphadenopathy (Cho-Vega et al., 2008; Magro et al., 2006; Staber et al., 2019). A recently published consensus classifies T-PLL into two phases (inactive and active) according to the clinical signs and progression, which demonstrate initially stable or slowly progressive disease followed by an active (acute) phase within 1–2 years (Staber et al., 2019). Although it is not exactly matched, considering the clinical course of this case, it can be considered similar to the inactive phase of T-PLL. The incidence of skin involvement in T-PLL ranges from 25% to 30% in human (Cho-Vega et al., 2008). Cutaneous lesions present with swelling and erythematous and/or purpuric lesions; due to red blood cell extravasation. Skin infiltrates usually involve the upper dermis with a perivascular and periadnexal distribution, and they rarely form a subcutaneous mass (Cho-Vega et al., 2008). One of the main clinical presentations is ecchymosis, which can be caused by vasculitis. Injury of the vessels may be due to direct or indirect sensitisation of malignant cells or due to cytokines released from leukaemic cells. Thus, LC should be considered when multifocal purpura and ecchymosis are observed in CLL patients. In addition to coagulation tests, histopathological analysis, including immunohistochemistry, should be conducted to rule out LC.

Vasculitis commonly occurs in human leukaemia, often as a reactive process caused by infections, drugs, or the malignancy itself (Cañueto et al., 2011). It involves direct destruction of the vessel walls by leukaemic cells (Cañueto et al., 2011). Histopathologically, the infiltration of the vessel walls and perivascular lesions by malignant cells, coupled with fibrin deposition and focal necrosis of these vessels, are observed (Çabuk et al., 2004; Cañueto et al., 2011). The underlying pathogenesis of skin migration of leukaemic cells is unclear in human LC but may involve a combined expression of different chemokine receptors and specific adhesion molecule receptors (Cho-Vega et al., 2008).

CD5-positive leukaemic B cells may be involved in the pathogenesis of vasculitis in humans (Hamidou et al., 2001). Most cases of T-PLL are positive for CD3, CD4, CD45RO, and T-cell leukaemia-1 and negative for CD2a, CD34, and TdT. CD4 and CD8 are co-expressed in 25% of cases, but rarely, both are absent (Matutes, 1998). In the current case, the immunophenotype was CD3+/CD5+/CD4+/-CD8+/-CD21+/-CD79a+/CD34-. Cytologically, T-PLL cells in tissue sections are slightly larger than normal lymphocytes and frequently have relatively round nuclear contours and prominent nucleoli (Cho-Vega et al., 2008). In our case, the small- to intermediate-sized lymphocytes had round hyperchromatic nuclei, indistinct nucleoli, and small to moderate amounts of cytoplasm.

**FIGURE 3** (a) The superficial dermis shows lichenoid infiltration of neoplastic lymphocytes (asterisk) in a perivascular to interstitial pattern. No epitheliotropism is noted. (b) The infiltrated lymphocytes (asterisk) showed mild nuclear and cytoplasmic pleomorphism, with rare mitotic figures. The neoplastic monomorphic lymphocytes are (c) strongly positive for CD3 on immunohistochemistry and negative for CD79a (d).
The clinical presentation of pre-T-ALL is often one or multiple cutaneous nodular lesions, and the leukaemia cells are positive for CD34 (a haematopoietic stem cell marker). However, our patient had no nodular lesions and CD34 stained negative.

ATLL is associated with human T-cell lymphotropic virus type I, and the lesion shows epitheliotropism with Pautrier’s microabscesses. However, in this case, lymphocyte infiltrates were mostly localised to the superficial dermis, and no intraepidermal pustules of lymphocytes were observed.

Our case has a different clinical course from mycosis fungoides (MF), which is a cutaneous primary T-cell lymphoma, and SS, which is an aggressive form of MF that constitutes a tumour in the peripheral lymph nodes. In general, MF mainly shows skin lesions, and it shows a systemic form in the later stages as the disease stage progresses (Fontaine et al., 2010). However, it is difficult to differentiate the lesion from other skin lesions solely based on gross appearance and clinical course of the lesion. In our case, since CLL was diagnosed 8 months before the appearance of the skin lesion, the possibility of primary skin tumours is low. In addition, histologically, MF and SS show epitheliotrophic features, whereas in this patient, neoplastic lymphocytes were confined to the superficial dermis and did not infiltrate into the epithelium. More studies based on different immunophenotypes of canine leukaemia should be conducted in the future.

Several studies have indicated that the presence of LC in patients with myeloid leukaemia or acute lymphoblastic leukaemia indicates a poor prognosis with a short survival time (Cho-Vega et al., 2008), probably due to the malignant nature of B-CLL. However, LC in the form of small lymphocytic infiltrations in human CLL patients is known to have a good prognosis that does not significantly affect patients’ survival (Cerroni et al., 1996; Colburn et al., 2002). This may also be true in dogs, as the CLL in this case was well-controlled after treatment with chemotherapeutic agents, and no skin lesions were observed.

This case report describes a rare case of LC in a dog with chronic T-cell lymphocytic leukaemia. When clinical signs of multifocal purpura and ecchymosis are observed in CLL patients, histopathological analysis, including immunohistochemistry, should be considered to rule out LC.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

ETHICS STATEMENT

Protection of human subjects and animals in research are not applicable in this study.

AUTHOR CONTRIBUTIONS

Hyeona Bae, Ji-Seon Yoon, Dae Young Kim, and DoHyeon Yu contributed to conception and design of the study. Eulsoo Choi, Sang-Hyun Kim, Dong-In Jung, Jinho Park, and Sang-Ki Kim organised the database. Hyeona Bae and Ji-Seon Yoon wrote the first draft of the manuscript. Eulsoo Choi, Sang-Hyun Kim, Dong-In Jung, Jinho Park, Sang-Ki Kim, Dae Young Kim, and DoHyeon Yu wrote sections of the manuscript. All authors contributed to manuscript revision, read, and approved the submitted version.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

PEER REVIEW

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