

# Hematological parameters in dogs at the early stages of babesiosis in the Dnipro region of Ukraine

A. Yu. Nevidnyk-Pravda, G. O. Ushakova aaasssaaa079@gmail.com



Oles Honchar Dnipro National University, 72 Naukovy Ave., Dnipro, 49010, Ukraine

#### ORCID:

A. Yu. Nevidnyk-Pravda https://orcid.org/0009-0002-8622-7566

G. O. Ushakova https://orcid.org/0000-0002-5633-2739

#### **Authors' Contributions:**

**NPAY:** Conceptualization; Methodology; Investigation; Data curation; Formal analysis; Writing — original draft; Visualization.

**UGO:** Supervision; Project administration; Writing — review & editing; Resources; Validation.

### Declaration of Conflict of Interests:

None to declare.

#### Ethical approval:

The experiments were carried out in accordance with the protocols approved by the local Ethics Committee (Peredovyi Veterinary Complex, Dnipro, Ukraine) and with the main provisions of the Law of Ukraine "On the Protection of Animals Against Cruelty" (no. 3447-IV from 21.02.2006) and "The European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes" (Strasbourg, 1986).

#### Acknowledgements:

None.



Attribution 4.0 International (CC BY 4.0)

**Key words:** dogs, blood, hematological parameters, *Babesia canis*, erythrocyte, leukocyte, anaemia, differential diagnostics

Canine babesiosis is a vector-borne disease caused by pro-

tozoan parasites of the genus *Babesia*, primarily transmitted by

ixodid ticks. The disease is widespread globally, including across

most regions of Ukraine. *Babesia canis* is the most prevalent species affecting dogs, with increasing clinical relevance in both domestic and wild carnivores. Infection leads to intravascular hemolysis, hypoxic tissue injury, and multi-organ dysfunction. Despite advances in diagnostics, early hematological changes in

the initial stages of infection remain underexplored. This study

aims to compare the hematological parameters of dogs in the

early stage of babesiosis with healthy controls to identify reliable

indicators for early diagnosis and disease monitoring. This study was conducted on 13 clinical cases of *Babesia canis* infection in dogs, with data collected directly from animals presented at the Peredovyi Veterinary Complex (Dnipro, Ukraine) between February and April 2024. Blood smears confirmed parasitemia, and

complete blood counts were performed using the *MicroCC-20 Plus* automated analyzer. Results showed a statistically significant decrease in red blood cell count (3.59±0.37×10<sup>6</sup>/µL) and hemoglobin (83.42±2.96 g/L) in infected dogs compared to controls (6.36±0.17×10<sup>6</sup>/µL and 158.58±5.87 g/L, respectively).

Hematocrit values were also markedly reduced (22.54±1.45 %

vs. 43.51±2.39 %; P<0.0001). Significant thrombocytopenia

(38.23±6.20×10<sup>3</sup>/µL) and leukopenia (7.08±0.60×10<sup>9</sup>/L) were

observed, with a concurrent neutrophilic shift and lymphopenia.

Mean corpuscular volume (MCV) was significantly lower in

the infected group (63.45±2.49 fL), while other red cell indices

(MCHC, RDW) and total protein levels showed no statistically

significant differences. These findings highlight the pronounced hematological disturbances associated with early-stage *Babesia canis* infection. The changes in erythrocyte count, hemoglobin concentration, hematocrit, and platelet levels may serve as early

diagnostic markers. Further research is needed to refine hemato-

logical profiling for improved clinical decision-making and timely

intervention in canine babesiosis.

#### Introduction

Babesiosis is an infectious natural focal disease caused by unicellular parasites of the genus *Babesia*, which belong to the class of sporozoites (*Apicomplexa*)

[28]. These parasites are intracellular hemoparasites that infect erythrocytes of mammals, including dogs, cats, wild carnivores and, less commonly, humans [6, 43, 50]. Infection usually occurs through the bites of *Ixodes* ticks (family *Ixodidae*), which are natural vectors of the pathogen [13].

The geographical distribution of babesiosis covers most temperate and tropical regions of the world, including the territories of Ukraine, where the disease is endemic [41]. In recent decades, there has been an increase in the incidence of the disease in animals due to changes in climatic conditions, migration of vectors and expansion of tick ranges [15, 16, 56]. Large-scale monitoring studies have shown the spread of *Dermacentor reticulatus* in Central and Eastern Europe, including Germany, Poland and the Czech Republic, which significantly increases the risk of canine infection [13, 17, 18, 42, 47, 49, 57]. In southeastern and northeastern Europe, babesiosis is considered an emerging and re-emerging disease, which emphasizes its growing epidemiological significance [3, 4, 20].

The pathogenesis of babesiosis is associated with the penetration of parasites into erythrocytes, where they undergo several cycles of division, causing their destruction (hemolysis) [26; 27; 31; 59]. This leads to the development of hemolytic anemia, accompanied by fever, jaundice, weakness, and multiple organ failure in severe cases [8, 35]. Mortality in virulent forms of canine babesiosis is often linked to consumptive coagulopathy and systemic inflammatory responses [5, 26]. In addition, alterations in hemostasis and coagulation markers have been confirmed as important prognostic indicators of disease progression [7, 35].

The immune response plays a decisive role in the clinical course of babesiosis. Cytokine-mediated inflammation and immune exhaustion mechanisms, similar to those observed in malaria, are implicated in the severity of the disease [36, 58]. Moreover, reinfections occur frequently, since post-infectious immunity is short-lived and often non-sterile [12].

The clinical manifestations of babesiosis are highly variable and depend on the type of parasite, the degree of parasitemia, the immune status of the animal, and the presence of concomitant infections (e.g., Ehrlichia canis) [14, 21, 22, 50]. Thus, two main species of Babesia are most commonly found in dogs: B. canis and B. gibsoni, which differ in size, pathogenesis, and sensitivity to therapy [1, 6]. Molecular epidemiological studies confirm a high genetic diversity of B. canis, which may complicate diagnosis and therapy [29]. Global distribution studies also indicate regional differences in species prevalence and tick associations [9].

Diagnosis of babesiosis includes both traditional methods and modern molecular tests. Microscopic examination of blood smears is a rapid and accessible method, but has low sensitivity at low parasitemia levels [33]. Enzyme-linked immunosorbent assays (ELISA) and immunofluorescence tests can detect antibodies, but they do not always distinguish between active and past infections [24, 34]. The most accurate method is the polymerase chain reaction (PCR), which can detect parasite DNA and identify its species [1, 10, 16]. Novel PCR-based assays have been successfully used for rapid field diagnostics and species differentiation [32, 34].

Treatment of babesiosis is based on the use of antiparasitic drugs, among which imidocarb dipropionate and atovaquone are the most effective [2, 12]. However, the choice of therapy depends on the type of pathogen and the severity of the disease. Supportive therapy is often essential to manage anemia, oxidative stress, and systemic inflammation [25, 53]. Clinical outcomes are strongly associated with early therapeutic intervention and the ability to correct hematological abnormalities [46, 51].

Hematological changes are of particular diagnostic and prognostic importance. Numerous studies have demonstrated alterations in erythrocytic and platelet indices during infection, which can serve as early diagnostic markers [25, 55, 61]. Severe thrombocytopenia and coagulation disorders are especially characteristic in acute babesiosis [19, 31]. Additionally, microcirculatory disturbances and cardiovascular complications are increasingly recognized as causes of mortality in infected dogs [8, 60].

Thus, babesiosis in animals and humans is an important problem of veterinary and medical parasitology, requiring an integrated approach to ensure effective control and treatment. The study of hematological changes in the early stages of babesiosis is of particular relevance, as it allows timely diagnosis and the identification of prognostic markers of disease severity.

The aim of this study is to perform a comparative analysis of hematological parameters in dogs in the early stages of babesiosis to identify diagnostically significant changes.

#### **Materials and Methods**

This study was conducted on 13 dogs presenting with the first signs of *Babesia canis* infection at the Peredovyi Veterinary Complex (Dnipro, Ukraine) between February and April 2024. All clinical and laboratory data were collected prospectively by the authors, including blood sampling, smear preparation, and hematological analysis. The study included dogs of various breeds, ages, and sexes. Although these factors may influence hematological parameters, the primary focus was on early-stage *Babesia canis* infection. Statistical analysis was conducted to compare infected animals with healthy controls, minimizing potential confounding effects of breed, age, and sex. Dogs with diagnosed concomitant diseases or with unreliable laboratory data were excluded from the study to ensure data reliability.

Detection of *Babesia canis* parasites in erythrocytes was carried out using thin blood smears stained with fast-acting dyes *LEUCODIF 200* (*Erba Lachema*, Czech Republic), with subsequent examination under 100x magnification using an optical microscope *Leica DM4* (Germany) (fig. 1).

The stage of babesiosis in dogs was determined based on a combination of clinical signs and laboratory parameters. The early stage included animals that showed mild lethargy, loss of appetite, moderate fever, pale or icteric mucous membranes, sometimes dark urine, but without pronounced anemia or intoxication. Laboratory tests at the early stage revealed 1 infected

erythrocyte per 10–15 fields of view in the study of blood smears, confirming a low level of parasitemia. Only the early stage of the disease was assessed in this study; therefore, differentiation between intermediate and late stages was not performed.

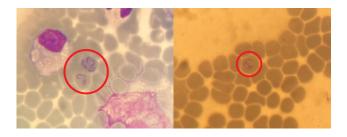
For hematological studies, blood was taken from the cephalic or subcutaneous vein into a tube with EDTA. After that, parameters such as the number of erythrocytes, hemoglobin level, leukocytes, platelets, mean hemoglobin concentration in erythrocytes, erythrocyte distribution width, total protein and hematocrit were analyzed. The analysis of these parameters was carried out using an automatic hematological analyzer *MicroCC-20 Plus (HTI*, USA). Quantitative assessment of segmented neutrophils and lymphocytes was carried out by microscopic counting on stained blood smears.

Animal handling complied with the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986) and the Regulations on the Use of Animals in Biomedical Research. All procedures were performed in accordance with ethical standards for working with experimental animals and were approved by the local Ethics Committee.

A variety of statistical methods were used to study hemolytic anemia in dogs caused by the protozoan parasite *Babesia canis*. Descriptive statistics were used to determine the mean, median, mode, standard deviation, and variance of the indicators in the control group and the group of animals with the initial stage of the disease. The *t*-test was used to compare the mean values. Additionally, analysis of variance (ANOVA) was used to compare the mean values in the groups.

#### **Results and Discussion**

Key hematological parameters were analyzed in 13 clinically healthy dogs (control group) and 13 dogs showing signs of the initial stage of babesiosis.



**Fig. 1.** Blood smear of a dog with *Babesia canis* detected (Leucodiff stain, ×100 oil immersion, NA 1.25). Intraerythrocytic forms of the parasite are visible (highlighted with a red circle)

The average RBC count in the control group (fig. 2) was  $6.36\pm0.17\times10^6/\mu$ L, whereas in the infected dogs, the count decreased significantly to  $3.59\pm0.37\times10^6/\mu$ L (P<0.0001; F=0.0107). This substantial reduction indicates a pronounced anemia likely caused by parasite-induced hemolysis.

Similarly, hemoglobin levels in the control dogs averaged 158.58±5.87 g/L, while in the infected group, HGB dropped markedly to 83.42±2.96 g/L (P<0.0001; F=0.0250). The decline reflects impaired oxygen transport capacity and suggests severe erythrocyte destruction during the course of infection. A parallel trend was observed in hematocrit values. Control animals showed a mean HCT of 43.51±2.39 %, which fell dramatically to 22.54±1.45 % in the infected group (P<0.0001; F=0.0955). The significant differences in all three parameters between healthy and infected dogs underscore the profound hematological disruption caused by *Babesia* infection prior to any therapeutic intervention.

The mean total protein level (fig. 3) in the control group was 67.42±3.36 g/L. In contrast, infected dogs exhibited a slightly lower mean value of 65.59±2.26 g/L.

Although the reduction was not statistically significant (P=0.1300; F=0.1845), the trend suggests a possible mild disturbance in protein metabolism or plasma volume shifts associated with the acute phase of infection.

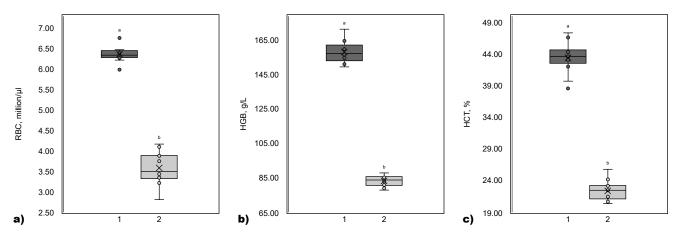


Fig. 2. Red blood cell count (a), hemoglobin concentration (b), and hematocrit (c) in the blood of dogs infected with Babesia canis compared to the control group (x±SD, n=13)

Note. Here and in the next figures 1 — control group of healthy dogs; 2 — group of dogs infected with Babesia canis.

 $^{\rm a}$ ,  $^{\rm b}$  — mean values with unlike letters were significantly different between the groups (P<0.0001).

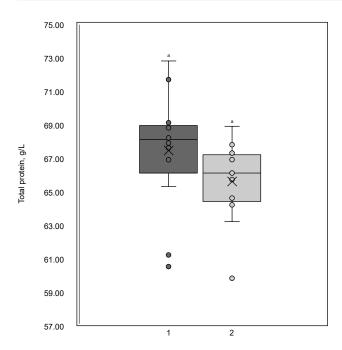


Fig. 3. Total protein concentration in the blood serum of dogs infected with *Babesia canis* and control group (x±SD, n=13)

The MCV value in the control group (fig. 4) averaged 66.38±3.13 fL, while in infected dogs, MCV was significantly lower at 63.45±2.49 fL (P=0.0178; F=0.4335). This reduction may reflect the predominance of microcytic erythrocytes or early regenerative responses during the course of infection.

MCHC values remained relatively stable between the groups. The control cohort had a mean MCHC of 362.56±9.56 g/L, compared to 362.09±5.58 g/L in the infected group (P=0.8844; F=0.0739), indicating no statistically significant difference. This suggests that hemoglobin concentration within individual red blood cells was largely unaffected by the infection at this stage. Similarly, RDW values showed only a slight, non-significant change.

The control group had a mean RDW of  $16.15\pm1.03~\%$ , while the infected group presented a mean of  $15.99\pm1.04~\%$ 

(P=0.7061; F=0.9790). The minimal variation suggests that anisocytosis (variation in red blood cell size) was not markedly increased in the early stages of *Babesia* infection.

The mean PLT count in the control group (fig. 5) was  $330.23\pm26.44\times10^3/\mu$ L. In contrast, dogs with *Babesia* infection exhibited a profound and statistically significant reduction in platelet levels, averaging just  $38.23\pm6.20\times10^3/\mu$ L (P<0.0001; F<0.0001). This sharp decline clearly indicates the presence of severe thrombocytopenia, a common clinical manifestation of canine babesiosis.

In the control group, the mean WBC count (fig. 6) was 11.29±1.04×10°/L, whereas in dogs with babesiosis, it decreased significantly to 7.08±0.60×10°/L (P<0.0001), indicating the presence of leukopenia, which is characteristic of the acute phase of the disease.

The relative count of segmented neutrophils was moderately increased in the diseased group (60.66±1.88 %) compared to the control group (57.15±2.82 %) (P=0.0015), suggesting a neutrophilic shift typical of acute inflammatory responses.

Conversely, the relative count of lymphocytes was significantly reduced in the *Babesia canis*-infected group (28.45±1.56 %) compared to the healthy control group (30.45±1.65 %) (P=0.0056), which is consistent with a stress leukogram commonly observed during severe infectious processes.

Our findings align with previous studies that highlight significant hematological alterations during the early stages of canine babesiosis. One of the key parameters we observed was leukopenia, particularly a reduction in total white blood cell (WBC) count. This is consistent with the results of Eichenberger et al. [19], who reported that nonsurviving dogs with *Babesia canis* infection exhibited moderate leukopenia, while survivors generally maintained WBC levels within the reference range. Notably, leukopenia was also observed in up to 60 % of mild cases [37], suggesting that it is a sensitive though not entirely specific marker of disease severity. Similar diagnostic

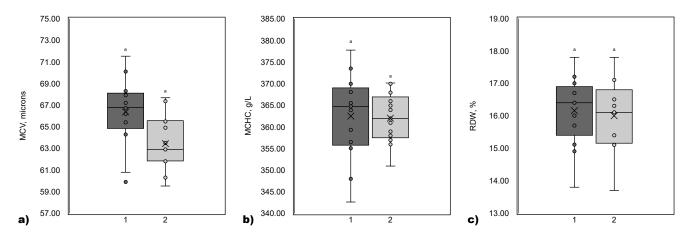


Fig. 4. Mean corpuscular volume (MCV) (a), mean corpuscular hemoglobin concentration (MCHC) (b), and red cell distribution width (RDW) (c) in dogs infected with Babesia canis compared to the control group (x±SD, n=13)

trends have been confirmed in cattle and small ruminants infected with different *Babesia* species, underscoring the conserved hematological response across hosts [1, 2].

Our data suggest that lymphopenia, especially during the early phase, may serve as an early indicator of immune suppression or immune dysregulation. This is in line with studies on *Babesia rossi*, where elevated cortisol levels were associated with immunosuppressive states and poor outcomes [44, 48]. Similarly, in malaria infections caused by *Plasmodium* spp., lymphocyte depletion has been attributed to redistribution, immune cell exhaustion, or parasite-induced apoptosis [30, 39, 58]. Immunological studies further suggest that post-infection immunity in dogs is often short-lived and non-sterile, which may explain why lymphocyte recovery is delayed [12]. These mechanisms may also contribute to the lymphopenia observed in *Babesia* infections.

Another critical hematological abnormality identified was thrombocytopenia, which was both profound and consistent in affected dogs. Thrombocytopenia is considered the most dramatic hematological change in babesiosis [25, 61]. Eichenberger et al. [19] proposed a prognostic cut-off of 27,500 platelets/µL, which, although not perfectly sensitive or specific, may still aid in early clinical decision-making. The pathogenesis of thrombocytopenia is likely multifactorial, involving systemic inflammatory responses (SIRS), platelet consumption, sequestration, and impaired production [7; 45]. Reports from endemic regions indicate that thrombocytopenia often precedes anemia, making it a valuable early diagnostic marker for clinicians [16].

Interestingly, although hemolytic anemia was expected, it was not always severe in the early stages of infection in our study. This finding is consistent with previous reports suggesting that anemia may develop progressively, depending on the stage of the disease and the balance between erythrocyte destruction and regeneration [35, 61]. This progressive anemia has also been linked to parasite genetic variability, which influences virulence and the rate of erythrocyte destruction [29].

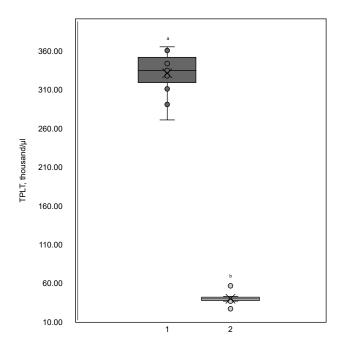
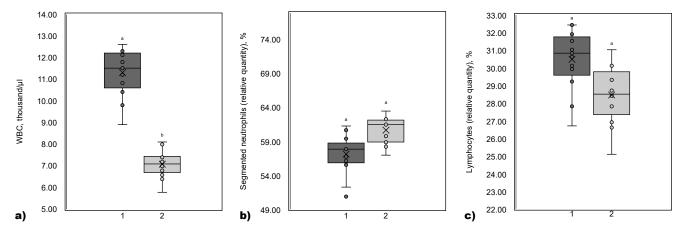


Fig. 5. Platelet (PLT) count in dogs infected with *Babesia canis* compared to the control group (x ± SD, n=13)

The hematologic responses observed in our study align with the findings of Scheepers et al. [46], who conducted a longitudinal analysis of transfused and non-transfused dogs naturally infected with Babesia rossi. Their work confirmed the presence of mild to moderate normocytic, normochromic regenerative anemia in all cases, consistent with the hemolytic nature of babesiosis. Notably, although transfusions effectively corrected anemia, they did not appear to significantly influence leukocyte or platelet dynamics, suggesting that the underlying pathophysiological mechanisms of inflammation and thrombocytopenia are independent of red blood cell restoration. This agrees with earlier observations that hematologic imbalances in babesiosis are driven more by immune-mediated and inflammatory mechanisms than by anemia alone [6].



**Fig. 6.** Total white blood cell (WBC) count (a), relative segmented neutrophil percentage (b), and relative lymphocyte percentage (c) in dogs infected with *Babesia canis* compared to the control group (x±SD, n=13)

Moreover, their observation of an inflammatory leukogram with a left shift, even in the absence of neutrophilia, highlights the atypical white cell responses in babesiosis and may reflect bone marrow suppression or consumption of neutrophils in peripheral tissues. Comparable atypical leukogram patterns have also been reported in *Babesia canis* and *Babesia gibsoni* infections, underscoring the complexity of immune responses during parasitemia [11]. The consistently severe thrombocytopenia, resolving within a few days, further supports the hypothesis of an immune-mediated etiology, rather than direct destruction by the parasite or marrow suppression.

These findings, when integrated with our data, strengthen the understanding that hematologic abnormalities such as anemia and thrombocytopenia are multifactorial in origin and dynamically evolve during the course of babesiosis. Early monitoring and interpretation of these patterns can help guide clinical decision-making, particularly regarding the need for transfusion and prognosis estimation. This is consistent with broader diagnostic recommendations emphasizing early hematologic surveillance in vector-borne diseases [1, 2].

Our findings are further supported by a large-scale retrospective analysis conducted by Fabisiak et al. [25], which statistically examined hematological abnormalities in 350 dogs diagnosed with Babesia spp. infection. Their results reinforce that thrombocytopenia is the most consistent and significant hematologic alteration in canine babesiosis, corroborating observations from our study and previous reports. Interestingly, their analysis also highlighted age- and breed-related variations in hematologic responses, including significant differences in PCV between young and adult dogs, as well as in total leukocyte counts between German Shepherds and mixed-breed dogs. Regional studies from Latin America and the Caribbean also suggest that epidemiological context may influence hematologic presentation, pointing to possible interactions between environmental and host-related factors [23].

These findings suggest that host factors such as age and breed may influence the severity of hematologic abnormalities, potentially affecting disease progression and prognosis. While our study did not stratify dogs by age or breed, the consistency of thrombocytopenia and the presence of varying degrees of anemia and leukopenia align with the broader population trends observed by Fabisiak et al. [25]. The rare but notable occurrence of bior pancytopenia in their dataset also warrants attention, particularly in severe or complicated cases of babesiosis. Notably, pancytopenia has also been observed in cases where parasitemia co-occurs with secondary infections, further complicating prognosis [12, 16].

Taken together, these insights highlight the multifactorial nature of hematologic responses in canine babesiosis, driven by parasite virulence, host immune status, and possibly genetic predispositions. Future studies incorporating larger sample sizes and breed-specific analysis may offer a more nuanced understanding of hematologic alterations and improve early prognostic capabilities [32].

Furthermore, exploring cytokine-driven mechanisms of anemia and thrombocytopenia may shed light on host–parasite interactions and provide novel therapeutic targets [29].

The study by Žvorc et al. [61] further complements our findings by providing a detailed assessment of erythrocyte and platelet indices in dogs naturally infected with large *Babesia*. Their results confirm that thrombocytopenia remains a consistent hematological hallmark, accompanied by a decrease in plateletcrit (PCT) and an increase in mean platelet volume (MPV), indicating platelet activation and consumption, possibly as part of a systemic inflammatory or coagulopathic process. Similar alterations in platelet indices have been noted in other vector-borne diseases, reinforcing the diagnostic relevance of MPV and PCT monitoring [6, 16]. These changes are consistent with the concept of immune-mediated thrombocytopenia or disseminated intravascular coagulation, as observed in other studies.

Moreover, the authors observed decreased RBC count, MCV, and hematocrit values both before and after treatment, which is consistent with the normocytic, normochromic anemia frequently seen in babesiosis. Interestingly, red cell distribution width (RDW) remained unchanged, suggesting a uniform population of erythrocytes and possibly limited regenerative response in many cases, which may reflect either the early stage of infection or a suppressed erythropoiesis due to systemic inflammation. Comparable patterns of anemia with poor regenerative response have also been described in *Babesia rossi* infections, highlighting the role of systemic inflammatory mediators in inhibiting bone marrow function [11, 29, 52].

These findings support the diagnostic and prognostic relevance of automated erythrocyte and platelet indices in canine babesiosis. Monitoring MPV and PCT in particular may offer insight into the pathophysiological processes underlying thrombocytopenia and help assess treatment efficacy and disease progression, especially when paired with classical parameters such as HCT and RBC count. As our study also indicated significant shifts in platelet and red cell parameters in affected animals, the integration of such indices could enhance early detection and prognostic stratification in clinical settings. Such integrative diagnostic approaches are strongly recommended in current guidelines for canine vector-borne diseases [1, 2].

This study confirms that canine babesiosis caused by *Babesia canis* leads to significant hematological alterations, including consistent thrombocytopenia, variable degrees of anemia, leukopenia, and lymphopenia, which can serve as valuable early diagnostic and prognostic markers. Thrombocytopenia, in particular, emerged as the most consistent and severe abnormality, likely resulting from immune-mediated mechanisms and systemic inflammation. Although anemia was common, it was often mild to moderate in early stages, suggesting progressive red blood cell destruction rather than acute hemolysis. Regional analyses also indicate that differences in prevalence and hematologic severity may be shaped by environmental and epidemiological factors [23, 38].

Our findings align with previous studies and emphasize the importance of monitoring platelet and erythrocyte indices, such as mean platelet volume (MPV), plateletcrit (PCT), and red blood cell parameters, to better understand disease progression and guide clinical decisions. The presence of leukopenia and lymphopenia also highlights possible immune dysregulation during infection, further supporting the need for timely intervention. Immunological studies suggest that reinfection resistance in dogs is often incomplete, making early monitoring of hematologic patterns essential for long-term disease control [12, 40, 54].

Overall, routine hematological profiling remains a critical component in the diagnosis and management of canine babesiosis. Early recognition of key changes in blood parameters can aid in identifying high-risk patients, optimizing treatment strategies, and improving clinical outcomes. Further studies are warranted to explore long-term hematologic and immunologic responses, as well as the influence of host factors such as breed and age on disease severity and prognosis.

#### References

- Alvarez JA, Rojas C, Figueroa JV. Diagnostic tools for the identification of *Babesia* sp. in persistently infected cattle. *Pathogens*. 2019; 8: 143. DOI: 10.3390/pathogens8030143.
- Antunes S, Rosa C, Couto J, Ferrolho J, Domingos A. Deciphering Babesia-vector interactions. Front Cell Infect Microbiol. 2017; 7: 429. DOI: 10.3389/fcimb.2017.00429.
- Bajer A, Beck A, Beck R, Behnke JM, Dwużnik-Szarek D, Eichenberger RM, Farkas R, Fuehrer HP, Heddergott M, Jokelainen P, Leschnik M, Oborina V, Paulauskas A, Radzijevskaja J, Ranka R, Schnyder M, Springer A, Strube C, Tolkacz K, Walochnik J. Babesiosis in southeastern, central and northeastern Europe: An emerging and re-emerging tick-borne disease of humans and animals. *Microorganisms*. 2022; 10 (5): 945. DOI: 10.3390/microorganisms10050945.
- Bajer A, Kowalec M, Levytska VA, Mierzejewska EJ, Alsarraf M, Poliukhovych V, Rodo A, Wężyk D, Dwużnik-Szarek D. Tick-borne pathogens, *Babesia* spp. and *Borrelia burgdorferi* sl, in sled and companion dogs from central and north-eastern Europe. *Pathogens*. 2022; 11 (5): 499. DOI: 10.3390/pathogens11050499.
- Beletić A, Janjić F, Radaković M, Spariosu K, Francuski Andrić J, Chandrashekar R, Tyrrell P, Radonjić V, Balint B, Ajtić J, Kovačević Filipović M. Systemic inflammatory response syndrome in dogs with naturally infected with *Babesia canis*: Association with the parasite load and host factors. *Vet Parasitol*. 2021; 291: 109366. DOI: 10.1016/j.vetpar.2021.109366.
- Baneth G, Bourdeau P, Bourdoiseau G, Bowman D, Breitschwerdt E, Capelli G, Cardoso L, Dantas-Torres F, Day M, Dedet JP, Dobler G, Ferrer L, Irwin P, Kempf V, Kohn B, Lappin M, Little S, Maggi R, Miró G, Naucke T, Oliva G, Otranto D, Penzhorn B, Pfeffer M, Roura X, Sainz A, Shaw S, Shin S, Solano-Gallego L, Straubinger R, Traub R, Trees A, Truyen U, Demonceau T, Fitzgerald R, Gatti D, Hostetler J, Kilmer B, Krieger K, Mencke N, Mendão C, Mottier L, Pachnicke S, Rees B, Siebert S, Stanneck D, Tarancón Mingote M, von Simson C, Weston S. Vector-borne diseases — constant challenge for practicing veterinarians: Recommendations from the CVBD World Forum. *Parasites Vectors*. 2012; 5: 55. DOI: 10.1186/1756-3305-5-55.
- Barić Rafaj R, Kules J, Selanec J, Vrkić N, Zovko V, Zupančič M, Trampuš Bakija A, Matijatko V, Crnogaj M, Mrljak V. Markers of

- coagulation activation, endothelial stimulation, and inflammation in dogs with babesiosis. *J Vet Intern Med.* 2013; 27 (5): 1172–1178. DOI: 10.1111/jvim.12146.
- Bartnicki M, Łyp P, Dębiak P, Staniec M, Winiarczyk S, Buczek K, Adaszek Ł. Cardiac disorders in dogs infected with *Babesia canis*. Pol J Vet Sci. 2017; 20 (3): 573–581. DOI: 10.1515/pjvs-2017-0070.
- Birkenheuer AJ, Buch J, Beall MJ, Braff J, Chandrashekar R. Global distribution of canine *Babesia* species identified by a commercial diagnostic laboratory. *Vet Parasitol Reg Stud Rep.* 2020; 22: 100471. DOI: 10.1016/j.vprsr.2020.100471.
- Bilwal A, Mandali G, Tandel F. Liver enzyme activity in dogs infected with Babesia canis. Intas Polivet. 2018; 19 (II): 313–314. Available at: https://www.cabidigitallibrary.org/doi/pdf/10.5555/20193238376
- Boozer AL, Macintire DK. Canine babesiosis. Vet Clin N Am Small Anim Pract. 2003; 33 (4): 885–904. DOI: 10.1016/S0195-5616(03)00039-1.
- Brandão LP, Hagiwara MK, Myiashiro SI. Humoral immunity and reinfection resistance in dogs experimentally inoculated with *Babesia canis* and either treated or untreated with imidocarb dipropionate. *Vet Parasitol.* 2003; 114 (4): 253–265. DOI: 10.1016/S0304-4017(03)00130-4.
- Daněk O, Hrazdilová K, Kozderková D, Jirků D, Modrý D. The distribution of *Dermacentor reticulatus* in the Czech Republic re-assessed: Citizen science approach to understanding the current distribution of the *Babesia canis* vector. *Parasites Vectors*. 2022; 15: 132. DOI: 10.1186/s13071-022-05242-6.
- Dantas-Torres F. Biology and ecology of the brown dog tick, Rhipicephalus sanguineus. Parasites Vectors. 2010; 3: 26. DOI: 10.1186/1756-3305-3-26.
- Dantas-Torres F, Ketzis J, Mihalca AD, Baneth G, Otranto D, Tort GP, Watanabe M, Linh BK, Inpankaew T, Castro PDJ, Borrás P, Arumugam S, Penzhorn BL, Ybañez AP, Irwin P, Traub RJ. TroCCAP recommendations for the diagnosis, prevention and treatment of parasitic infections in dogs and cats in the tropics. *Vet Parasitol.* 2020; 283: 109167. DOI: 10.1016/j.vetpar.2020.109167.
- Djokic V, Rocha SC, Parveen N. Lessons learned for pathogenesis, immunology, and disease of erythrocytic parasites: *Plasmodium* and *Babesia*. Front Cell Infect Microbiol. 2021; 11: 685239.
  DOI: 10.3389/fcimb.2021.685239.
- Drehmann M, Springer A, Lindau A, Fachet K, Mai S, Thoma D, Schneider CR, Chitimia-Dobler L, Bröker M, Dobler G, Mackenstedt U, Strube C. The spatial distribution of *Dermacentor* ticks (*Ixodidae*) in Germany — evidence of a continuing spread of *Dermacentor reticulatus*. Front Vet Sci. 2020; 7: 578220. DOI: 10.3389/fvets.2020.578220.
- Dwużnik-Szarek D, Mierzejewska EJ, Rodo A, Goździk K, Behnke-Borowczyk J, Kiewra D, Kartawik N, Bajer A. Monitoring the expansion of *Dermacentor reticulatus* and occurrence of canine babesiosis in Poland in 2016–2018. *Parasites Vectors*. 2021; 14: 267. DOI: 10.1186/s13071-021-04758-7.
- Eichenberger RM, Riond B, Willi B, Hofmann-Lehmann R, Deplazes P. Prognostic markers in acute *Babesia canis* infections. *J Vet Intern Med*. 2016; 30 (1): 174–182. DOI: 10.1111/jvim.13822.
- Efstratiou A, Karanis G, Karanis P. Tick-borne pathogens and diseases in Greece. *Microorganisms*. 2021; 9 (8): 1732. DOI: 10.3390/microorganisms9081732.
- Eslahi AV, Mowlavi G, Houshmand E, Pirestani M, Majidiani H, Nahavandi KH, Johkool MG, Badri M. Occurrence of *Dioctophyme renale* (Goeze, 1782) in road-killed canids of Iran and its public health implication. *Vet Parasitol Reg Stud Rep.* 2021; 24: 100568. DOI: 10.1016/j.vprsr.2021.100568.
- Eslahi AV, Olfatifar M, Zaki L, Pirestani M, Sotoodeh S, Farahvash MA, Maleki A, Badri M. The worldwide prevalence of intestinal helminthic parasites among food handlers: A systematic review and meta-analysis. *Food Control*. 2023; 148: 109658. DOI: 10.1016/j.foodcont.2023.109658.
- Galon EM, Zafar I, Ji S, Li H, Ma Z, Xuan X. Molecular reports of ruminant *Babesia* in southeast Asia. *Pathogens*. 2022; 11 (8): 915. DOI: 10.3390/pathogens11080915.

- Garcia K, Weakley M, Do T, Mir S. Current and future molecular diagnostics of tick-borne diseases in cattle. *Vet Sci.* 2022; 9 (5): 241. DOI: 10.3390/vetsci9050241.
- Fabisiak M, Sapierzyński R, Kluciński W. Analysis of haematological abnormalities observed in dogs infected by a large Babesia. Bull Vet Inst Pulawy. 2010; 54 (2): 167–170. Available at: https://www.researchgate.net/publication/289677912\_Analyis\_of\_haematological\_abnormalities\_observed\_in\_dogs\_infected\_by\_a\_large\_Babesia
- Goddard A, Wiinberg B, Schoeman JP, Kristensen AT, Kjelgaard-Hansen M. Mortality in virulent canine babesiosis is associated with a consumptive coagulopathy. Vet J. 2013; 196 (2): 213–217. DOI: 10.1016/j.tvjl.2012.09.009.
- Jacobson LS, Clark IA. The pathophysiology of canine babesiosis: New approaches to an old puzzle. J S Afr Vet Assoc. 1994; 65 (3): 134–145. PMID: 7595923.
- Jalovecka M, Sojka D, Ascencio M, Schnittger L. Babesia life cycle — when phylogeny meets biology. *Trends Parasitol*. 2019; 35 (5): 356–368. DOI: 10.1016/j.pt.2019.01.007.
- Helm SC, Weingart C, Ramünke S, Schäfer I, Müller E, von Samson-Himmelstjerna G, Kohn B, Krücken J. High genetic diversity of *Babesia canis* (Piana & Galli-Valerio, 1895) in a recent local outbreak in Berlin/Brandenburg, Germany. *Transbound Emerg Dis.* 2022; 69: e3336. DOI: 10.1111/tbed.14617.
- Kassa D, Petros B, Mesele T, Hailu E, Wolday D. Characterization of peripheral blood lymphocyte subsets in patients with acute *Plas-modium falciparum* and *P. vivax* malaria infections at Wonji Sugar Estate, Ethiopia. *Clin. Vaccine Immunol.* 2006; 13 (3): 376–379. DOI: 10.1128/CVI.13.3.376-379.2006.
- Kettner F, Reyers F, Miller D. Thrombocytopaenia in canine babesiosis and its clinical usefulness. J S Afr Vet Assoc. 2003; 74 (3): 63–68. DOI: 10.4102/jsava.v74i3.512.
- Kirtz G, Leschnik M, Hooijberg E, Tichy A, Leidinger E. In-clinic laboratory diagnosis of canine babesiosis (*Babesia canis canis*) for veterinary practitioners in Central Europe. *Tierarztl Prax Ausg K Kleintiere Heimtiere*. 2012; 40 (2): 87–94. DOI: 10.1055/s-0038-1623628.
- 33. Kuleš J, Potocnakova L, Bhide K, Tomassone L, Fuehrer HP, Horvatić A, Galan A, Guillemin N, Nižić P, Mrljak V, Bhide M. The challenges and advances in diagnosis of vector-borne diseases: where do we stand? *Vector Borne Zoonotic Dis.* 2017; 17 (5): 285–296. DOI: 10.1089/vbz.2016.2074.
- 34. Kuo CY, Zhao C, Cheng T, Tsou CC, Li YC, Zhang Y, Hsieh MC, Haung SB, Chen WY. Rapid identification of *Babesia canis* and *Babesia gibsoni* (Asian genotype) in canine blood samples using a customized portable real-time PCR analyzer and TaqManbased assay. *Ticks Tick Borne Dis.* 2020; 11 (2): 101362. DOI: 10.1016/j.ttbdis.2019.101362.
- Liebenberg C, Goddard A, Wiinberg B, Kjelgaard-Hansen M, van der Merwe LL, Thompson PN, Matjila PT, Schoeman JP. Hemostatic abnormalities in uncomplicated babesiosis (*Babesia rossi*) in dogs. J Vet Intern Med. 2013; 27 (1): 150–156. DOI: 10.1111/jvim.12016.
- Leisewitz A, Goddard A, De Gier J, Van Engelshoven J, Clift S, Thompson P, Schoeman JP. Disease severity and blood cytokine concentrations in dogs with natural *Babesia rossi* infection. *Parasite Immunol*. 2019; 41 (7): e12630. DOI: 10.1111/pim.12630.
- Máthé A, Vörös K, Nemeth T, Biksi I, Hetyey C, Manczur F, Tekes L. Clinicopathological changes and effect of imidocarb therapy in dogs experimentally infected with *Babesia canis. Acta Vet Hung.* 2006; 54 (1): 19–33. DOI: 10.1556/avet.54.2006.1.3.
- Milanović Z, Beletić A, Vekić J, Zeljković A, Andrić N, Ilić Božović A, Spariosu K, Radaković M, Ajtić J, Kovačević Filipović M. Evidence of acute phase reaction in asymptomatic dogs naturally infected with *Babesia canis*. Vet Parasitol. 2020; 282: 109140. DOI: 10.1016/j.vetpar.2020.109140.
- Onishi T, Suzuki S, Horie M, Hashimoto M, Kajikawa T, Ohishi I, Ejima H. Serum hemolytic activity of *Babesia gibsoni-*infected dogs: the difference in the activity between self and nonself red blood cells. *J Vet Med Sci.* 1993; 55 (2): 203–206. DOI: 10.1292/jvms.55.203.

- 40. Otranto D, Dantas-Torres F, Fourie JJ, Lorusso V, Varloud M, Gradoni L, Drake J, Geurden T, Kaminsky R, Heckeroth AR, Schunack B, Pollmeier M, Beugnet F, Holdsworth P. World Association for the Advancement of Veterinary Parasitology (W.A.A.V.P.) guidelines for studies evaluating the efficacy of parasiticides in reducing the risk of vector-borne pathogen transmission in dogs and cats. *Vet Parasitol*. 2021; 290: 109369. DOI: 10.1016/j.vetpar.2021.109369.
- Panti-May JA, Rodríguez-Vivas RI. Canine babesiosis: A literature review of prevalence, distribution, and diagnosis in Latin America and the Caribbean. Vet Parasitol Reg Stud Rep. 2020; 21: 100417. DOI: 10.1016/j.vprsr.2020.100417.
- Pawełczyk O, Kotela D, Asman M, Witecka J, Wilhelmsson P, Bubel P, Solarz K. The first records of canine babesiosis in dogs from *Dermacentor reticulatus* — Free zone in Poland. *Pathogens*. 2022; 11 (11): 1329. DOI: 10.3390/pathogens11111329.
- Penzhorn BL. Don't let sleeping dogs lie: Unravelling the identity and taxonomy of *Babesia canis*, *Babesia rossi* and *Babesia vogeli*. *Parasites Vectors*. 2020; 13: 184. DOI: 10.1186/s13071-020-04062-w.
- 44. Penzhom BL, Harrison-White RF, Stoltsz WH. Completing the cycle: Haemaphysalis elliptica, the vector of Babesia rossi, is the most prevalent tick infesting black-backed jackals (Canis mesomelas), an indigenous reservoir host of B. rossi in South Africa. Ticks Tick Borne Dis. 2020; 11 (2): 101325. DOI: 10.1016/j.ttbdis.2019.101325.
- 45. Reyers F, Leisewitz AL, Lobetti RG, Milner RJ, Jacobson LS, van Zyl M. Canine babesiosis in South Africa: more than one disease. Does this serve as a model for falciparum malaria? *Ann Trop Med Parasitol*. 1998; 92 (4): 503–511. DOI: 10.1080/00034983.1998.11813308.
- Scheepers E, Leisewitz AL, Thompson PN, Christopher MM. Serial haematology results in transfused and non-transfused dogs naturally infected with Babesia rossi. *J S Afr Vet Assoc.* 2011; 82 (3): 136–143. DOI: 10.4102/jsava.v82i3.51.
- Schäfer I, Helm C, Marsboom C, Hendrickx G, Kohn B, Krücken J, Samson-Himmelstjerna G, Müller E. Infections with *Babesia* spp. in dogs living in Germany (2007–2020). *J Vet Intern Med*. 2021; 35: 3199.
- Schoeman JP, Herrtage ME. Adrenal response to the low dose ACTH stimulation test and the cortisol-to-adrenocorticotrophic hormone ratio in canine babesiosis. *Vet Parasitol*. 2008; 154 (3–4): 205–213. DOI: 10.1016/j.vetpar.2008.03.023.
- Seleznova M, Kivrane A, Namina A, Krumins R, Aleinikova D, Lazovska M, Akopjana S, Capligina V, Ranka R. Babesiosis in Latvian domestic dogs, 2016–2019. *Ticks Tick Borne Dis*. 2020; 11 (5): 101459. DOI: 10.1016/j.ttbdis.2020.101459.
- Solano-Gallego L, Sainz Á, Roura X, Estrada-Peña A, Miró G. A review of canine babesiosis: The European perspective. *Parasites Vectors*. 2016; 9: 336. DOI: 10.1186/s13071-016-1596-0.
- Strobl A, Künzel F, Tichy A, Leschnik M. Complications and risk factors regarding the outcomes of canine babesiosis in Central Europe — a retrospective analysis of 240 cases. *Acta Vet Hung*. 2020; 68 (2): 160–168. DOI: 10.1556/004.2020.00031.
- Sung LH, Sundaram AH, Glick AL, Chen DF, Shipton L. Babesiosis as a cause of atraumatic splenic injury: Two case reports and a review of literature. *J Gen Intern Med*. 2021; 36: 3869–3874. DOI: 10.1007/s11606-021-07117-5.
- Teodorowski O, Winiarczyk S, Tarhan D, Dokuzeylül B, Ercan AM, Or ME, Staniec M, Adaszek Ł. Antioxidant status, and blood zinc and copper concentrations in dogs with uncomplicated babesiosis due to *Babesia canis* infections. *J Vet Res.* 2021; 65 (2):169–171. DOI: 10.2478/jvetres-2021-0031.
- Thongsahuan S, Chethanond U, Wasiksiri S, Saechan V, Thongtako W, Musikacharoen T. Hematological profile of blood parasitic infected dogs in Southern Thailand. *Vet World*. 2020; 13 (11): 2388–2394. DOI: 10.14202/vetworld.2020.2388-2394.
- Tołkacz K, Rodo A, Wdowiarska A, Bajer A, Bednarska M. Impact of Babesia microti infection on the initiation and course of pregnancy in BALB/c mice. Parasites Vectors. 2021; 14: 132. DOI: 10.1186/ s13071-021-04638-0.

- Vannier E, Krause PJ. Babesiosis. In: Ryan ET, Hill DR, Solomon T, Aronson NE, Endy TP (eds.). Hunter's Tropical Medicine and Emerging Infectious Diseases. Elsevier, 2020. p. 799–802. DOI: 10.1016/B978-0-323-55512-8.00105-8.
- 57. Vatoliková I, Dekány D, Matušková H, Miklošovičová B, Macenauer Z, Szaboóvá A, Šimek J, Hanzlíček D. Babezióza psov na západnom Slovensku: retrospektívna klinická štúdia z rokov 2014–2018. *Veterinářství*. 2019; 69: 144–150.
- Wykes MN, Horne-Debets JM, Leow CY, Karunarathne DS. Malaria drives T cells to exhaustion. *Front Microbiol.* 2014; 5: 249. DOI: 10.3389/fmicb.2014.00249.
- Zygner W, Gójska-Zygner O, Norbury LJ. Pathogenesis of anemia in canine babesiosis: Possible contribution of pro-inflammatory cytokines and chemokines — a review. *Pathogens*. 2023; 12 (2): 166. DOI: 10.3390/pathogens12020166.
- Zygner W, Rodo A, Gójska-Zygner O, Górski P, Bartosik J, Kotomski G. Disorders in blood circulation as a probable cause of death in dogs infected with *Babesia canis*. *J Vet Res*. 2021; 65 (3): 277–285. DOI: 10.2478/jvetres-2021-0036.
- Žvorc Z, Baric Rafaj R, Kules J, Mrljak V. Erythrocyte and platelet indices in babesiosis of dogs. Vet. Arhiv. 2010; 80 (2): 259–267. Available at: https://wwwi.vef.hr/vetarhiv/papers/2010-80-2-10.pdf

## Гематологічні показники у собак на ранніх стадіях бабезіозу в Дніпропетровській області, Україна А. Ю. Невідник-Правда, Г. О. Ушакова аааsssaaa079@gmail.com

Дніпровський національний університет імені Олеся Гончара, проспект Науковий, 72, м. Дніпро, 49010, Україна

Бабезіоз собак — це трансмісивне захворювання, спричинене найпростішими паразитами роду Babesia, які передаються переважно через іксодових кліщів. Захворювання поширене в усьому світі, зокрема в більшості регіонів України. Babesia canis — найпоширеніший вид, який вражає собак, зі зростанням клінічного значення як для домашніх, так і для диких м'ясоїдних тварин. Інфекція призводить до внутрішньосудинного гемолізу, гіпоксичного пошкодження тканин та поліорганної дисфункції. Незважаючи на досягнення в діагностиці, ранні гематологічні зміни на початкових стадіях інфекції залишаються недостатньо вивченими. Метою цього дослідження є порівняти гематологічні параметри собак на ранніх стадіях бабезіозу зі здоровими контрольними тваринами, щоб визначити надійні показники для ранньої діагностики та моніторингу захворювання. Було проведено ретроспективний аналіз 13 клінічних випадків інфекції *Babesia canis* у собак, представлених на ветеринарному комплексі «Передовий» (Дніпро, Україна) у період з лютого по квітень 2024 р. Мазки крові підтвердили паразитемію, а загальний аналіз крові було проведено за допомогою автоматичного аналізатора MicroCC-20 Plus. Результати показали статистично вірогідне зниження кількості еритроцитів (3,59±0,37×10<sup>6</sup>/мкл) та гемоглобіну (83,42±2,96 г/л) у інфікованих собак, порівняно з контрольними тваринами (6,36±0,17×10<sup>6</sup>/мкл та 158,58±5,87 г/л відповідно). Значення гематокриту також були значно знижені (22,54±1,45 % проти 43,51±2,39 %; P<0,0001). Спостерігали значну тромбоцитопенію  $(38,23\pm6,20\times103/мкл)$  та лейкопенію  $(7,08\pm0,60\times10^9/л)$  з одночасним нейтрофільним зсувом та лімфопенією. Середній об'єм еритроцитів (MCV) в інфікованій групі був значно нижчим (63.45±2.49 фл), тоді як інші показники еритроцитів (MCHC, RDW) та рівень загального білка не показали статистично вірогідних відмінностей. Ці дані підкреслюють виражені гематологічні порушення, пов'язані з ранньою стадією інфекції Babesia canis. Зміни кількості еритроцитів, концентрації гемоглобіну, гематокриту та рівня тромбоцитів можуть слугувати ранніми діагностичними маркерами. Необхідні подальші дослідження для уточнення гематологічного профілювання, щоб покращити прийняття клінічних рішень та своєчасне втручання при бабезіозі собак.

**Ключові слова:** собаки, кров, гематологічні показники, *Babesia canis*, еритроцити, лейкоцити, анемія, диференціальна діагностика