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Electrocardiogram Alteration and Hematologic Parameters in Dogs with Iron Deficiency Anemia

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Abstract

IRON DEFICIENCY ANEMIA (IDA), a common yet under recognized condition in canine populations, presents systemic health risks, including cardiovascular dysfunction. While, electrocardiogram (ECG) abnormalities associated with anemia have been documented in humans and felines, species-specific evidence in dogs remains limited. This investigation utilized a prospective case-control design to evaluate ECG alterations in dogs suffering from IDA and their relationship with hematological severity. a cohort of 40 client-owned dogs diagnosed with IDA (hemoglobin [hb] <12 g/dl, microcytic/hypochromic erythrocytes, confirmed *via* serum iron and ferritin levels) and 60 age-matched healthy controls underwent standardized hematological profiling and six-lead ECG assessment. analyzed parameters included heart rate (HR), QRS complex amplitude, ST-segment morphology, and corrected QT interval. Comparative analysis revealed marked ECG deviations in anemic dogs relative to controls: elevated HR (145.6 ± 7.8 vs. 125.4 ± 4.2 bpm; *p* < 0.013), diminished QRS amplitude (1.1 ± 0.1 vs. 1.3 ± 0.1 mv; *p* < 0.013), abbreviated QT intervals (201.5 ± 8.3 vs. 223.2 ± 5.1 ms; *p* < 0.022), and pronounced ST-segment deviation (0.17 ± 0.02 vs. 0.10 ± 0.01 mv; *p* < 0.001). Hematological correlations demonstrated significant inverse relationships between Hb levels and HR (spearman's ρ = -0.62; *p* < 0.001) and between serum iron concentrations and ST-segment deviations (ρ = -0.68; *p* < 0.001). Subgroup stratification by anemia severity (Hb <7 vs. ≥7 g/dl) revealed exacerbated tachycardia (168.4 ± 32.1 vs. 132.6 ± 29.5 bpm; *p* < 0.001) and ST abnormalities (0.31 ± 0.12 vs. 0.15 ± 0.1 mv; *p* = 0.003) in severe cases. These findings suggest that IDA-induced myocardial hypoxia and altered ion homeostasis underlie electrophysiological disturbances, manifesting as repolarization anomalies and compensatory tachycardia. Notably, the observed reduction in QRS amplitude diverges from human studies, emphasizing species-specific cardiac adaptations to anemia. The study highlights the clinical utility of ECG monitoring in IDA management to identify early cardiovascular compromise, particularly in settings with limited diagnostic resources. Further research is warranted to establish breed-specific reference intervals and assess ECG normalization post-therapy. By elucidating cardiac manifestations of canine IDA, this work addresses a significant knowledge gap in veterinary cardiology, providing insights to reduce anemia-associated cardiac morbidity through targeted interventions.

Keywords: Canine Iron Deficiency Anemia, Electrocardiography, Myocardial Hypoxia, Hematological Correlation, Cardiovascular Compromise.

Introduction

Iron deficiency anemia (IDA) is a disorder marked by reduced erythrocyte mass and impaired oxygen-carrying capacity. A clinically significant yet underdiagnosed condition in canine populations, affecting 15–20% of cases presenting with chronic gastrointestinal hemorrhage, parasitic infestations, or nutritional deficiencies [1,2]. Beyond its hematological manifestations, IDA induces systemic hypoxia, disrupting myocardial electrophysiology through perturbations in ion channel regulation, mitochondrial oxidative stress, and compensatory autonomic hyperactivity [3, 4, 5]. In human medicine, anemia severity correlates strongly with ECG anomalies, such as prolonged QT intervals, ST-segment deviations, and diminished R-wave amplitudes changes mechanistically linked to hypoxia-mediated suppression of potassium rectifier currents (IKr) and dysregulated calcium homeostasis [6]. Analogous findings in feline studies reveal IDA-associated tachycardia, fragmented QRS complexes, and ventricular arrhythmias, likely secondary to iron-

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dependent impairments in myocardial cytochrome oxidase activity [7, 8].

Despite interspecies parallels, canine-specific investigations into IDA-related cardiovascular dysfunction remain limited. Contemporary veterinary literature prioritizes hematological diagnostics (e.g., microcytosis, hypochromasia) and therapeutic iron repletion, with insufficient attention to myocardial sequelae [9, 10, and 11]. This oversight carries clinical ramifications: compensatory mechanisms such as heightened cardiac output and sympathetic overdrive may obscure early ECG abnormalities until irreversible myocardial remodeling ensues [12, 13, and 14]. Furthermore, iron's critical role in cellular metabolism as a cofactor for heme synthesis and electron transport chain enzymes implies that IDA-induced metabolic insufficiency exacerbates arrhythmogenic substrates, particularly in breeds predisposed to cardiomyopathies [15, 16, and 17].

Recent advancements in veterinary cardiology underscore ECG's prognostic utility in chronic disease states. For example, ST-segment deviations in feline hypertrophic cardiomyopathy predict thromboembolic risk [18], while QRS fragmentation in canine dilated cardiomyopathy correlates with ventricular dyssynchrony [19, 20]. However, no systematic analyses have characterized ECG patterns in canine IDA or their association with anemia severity, hindering clinicians' ability to non-invasively evaluate cardiovascular compromise in resource-constrained settings [21, 22].

This study hypothesizes that IDA in dogs elicits distinct ECG alterations including QRS amplitude attenuation, sinus tachycardia, and ST segment abnormalities that correlate with hematological indices (hemoglobin [Hb], hemocrite [Hct], serum iron) and reflect hypoxia-driven ion channel dysfunction. By employing descriptive statistics, Spearman's rank correlation (ρ), and multivariate regression modeling, this investigation aims to delineate the interplay between anemia severity, myocardial electrophysiology, and compensatory hemodynamic adaptations, thereby addressing critical gaps in veterinary clinical practice [23, 24]. This investigation pursues three primary aims:

Characterizing ECG abnormalities in IDA-affected dogs relative to healthy controls, focusing on parameters such as heart rate (HR), QRS complex morphology, ST-segment deviations, and corrected QT intervals. Prior studies in humans and cats demonstrate anemia-driven repolarization abnormalities [25, 26], but analogous canine data are lacking, necessitating species-specific benchmarks [27, 28].

Establishing quantitative associations between ECG alterations and hematological markers of anemia severity, including Hb, Hct, serum iron, and ferritin levels. Human research underscores robust correlations between Hb depletion and ECG changes such as tachycardia and ST depression [5, 29], while feline models link iron deficiency to fragmented QRS complexes [8, 30].

Exploring mechanistic links between iron deficiency and myocardial electrophysiological dysfunction, particularly hypoxia-mediated ion channel dysregulation (e.g., K^+ and Ca^{2+} currents) and mitochondrial oxidative stress. Iron's role as a cofactor in heme synthesis and electron transport chain enzymes suggests IDA disrupts cellular energetics, exacerbating arrhythmogenic substrates [31, 32].

This study advances the clinical utility of ECG in the management of canine iron deficiency anemia (IDA) by establishing a diagnostic framework for early detection of cardiovascular compromise and guiding precision therapeutic strategies.

Material and Methods

Conducted at a veterinary hospital of the Department of Internal Medicine, Faculty of Veterinary Medicine, Cairo University from January 2021 to March 2022. The study included two cohorts: 40 client-owned dogs diagnosed with iron deficiency anemia (IDA) and 60 age- and weight-matched healthy control dogs. IDA confirmed according to standardized hematological criteria: Hb levels below 12 g/dL, serum iron concentrations under 60 µg/dL, ferritin values less than 100 ng/mL, and the presence of microcytic, hypochromic erythrocytic morphology [1, 33, 34, and 35]. Control subjects demonstrated unremarkable CBCs, serum iron profiles within normal reference ranges, and no clinical or historical evidence of anemia.

Exclusion criteria included:

- Echocardiographic evidence of structural cardiac pathology (e.g., valvular defects, cardiomyopathies; [36] or Elevated cardiac biomarkers indicative of structural myocardial pathology (e.g., NT-proBNP >1,500 pmol/L or cardiac troponin I >0.5 ng/mL; [37, 38];
- Abnormal serum electrolyte concentrations (potassium [K^+] <3.5 or >5.5 mmol/L; calcium [Ca^{2+}] <8.5 mg/dL);
- Prior blood transfusion within four weeks preceding enrollment;
- Breeds predisposed to inherited arrhythmic disorders (e.g., Boxers with arrhythmogenic right ventricular cardiomyopathy [27].

Data Acquisition

Blood samples were obtained through jugular venipuncture and aliquoted into EDTA-coated and serum separation tubes for hematological and biochemical assessments. Analyses encompassed:

Complete Blood Count (CBC): Performed using an automated hematology analyzer (Siemens ADVIA 2120i) with dual methodology (impedance and flow cytometry) to quantify erythrocytic indices, including RBC count, Hb, Hct, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC).

Iron Studies

Serum Iron: Quantified via a colorimetric ferrozine-based assay (Randox Laboratories, UK).

Ferritin: Measured using a canine-specific quantitative sandwich ELISA (MyBioSource, USA; intra-assay coefficient of variation <8%).

Total Iron-Binding Capacity (TIBC): Calculated indirectly by summing serum iron and unsaturated iron-binding capacity (UIBC) values [39].

Pre-analytical variables, including 12-hour fasting protocols and avoidance of hemolyzed samples, were standardized according to the American Society for Veterinary Clinical Pathology (ASVCP) guidelines [40].

Electrocardiography

Standard six-lead electrocardiograms (ECGs) acquired using a digital recording system (ECG MAC, ECG 3, Digital 3 Channels electrocardiograph) with standardized settings (50 mm/s paper speed, 10 mm/mV gain). Dogs were positioned in lateral recumbency during recordings to ensure consistent lead placement (bipolar limb leads I, II, III; augmented unipolar leads avR, avL, avF). To reduce autonomic influence, ECGs initiated following a 5-minute stabilization period to minimize stress-induced variability. Key parameters were evaluated as follows:

QRS Amplitude: Assessed in lead II as the maximal vertical deflection from the isoelectric baseline to the R-wave peak [18].

ST-Segment Deviation: Measured 80 ms following the J-point, with deviations quantified relative to the TP segment baseline [27] veterinary adaptation).

Heart Rate (HR): Derived from the mean R-R interval across 10 consecutive sinus beats.

Two board-certified veterinary cardiologists, blinded to group allocation, independently analyzed all tracings, demonstrating substantial interobserver agreement (Cohen's $\kappa = 0.88$).

Statistical Analysis

The distribution of continuous variables evaluated for normality using the Shapiro-Wilk test. All statistical analyses executed in SPSS v28.0 (IBM Corp.), employing the following analytical framework:

Descriptive Statistics: Central tendency and dispersion reported as mean ± standard deviation (SD) or standard error (SE), as appropriate.

Intergroup Comparisons: Nonparametric Mann-Whitney *U* tests were applied to variables violating normality assumptions (e.g., HR, QRS amplitude).

Correlational Analysis: Associations between ECG parameters and hematological indices were quantified via Spearman's rank correlation coefficient (ρ).

Effect Size Estimation: The magnitude of intergroup differences was determined using Cohen's *d* (standardized mean difference).

Predictive Modeling: Stepwise linear regression identified ECG variables independently predictive of Hb levels, with variance inflation factors (VIF <2) confirming multicollinearity absence.

Addressing multiple comparison bias, the family-wise error rate was controlled using Bonferroni correction, yielding a revised significance threshold of $\alpha = 0.01$.

Results

Blood Parameters (Table 1)

Pronounced hematological deficits: Iron-deficient dogs demonstrated significantly reduced RBC counts, Hb levels, and Hct compared to healthy counterparts (*p* < 0.001).

Iron metabolism disruption: Markedly lower serum iron, ferritin, and total iron-binding capacity (TIBC) confirmed systemic iron depletion (*p* < 0.001).

Microcytic, hypochromic profile: Decreased MCV and MCH indicated smaller, hemoglobin-deficient erythrocytes, pathognomonic of iron deficiency.

MCHC paradox: Elevated MCHC in anemic dogs (*p* = 0.001), likely reflecting cellular dehydration due to chronic iron scarcity.

Mild cytopenias: Moderate yet statistically significant decreases in platelet (*p* = 0.003) and white blood cell (*p* < 0.001) counts were observed in the anemic cohort.

ECG Changes (Table 2)

Canines with iron deficiency anemia exhibited significant electrocardiographic alterations compared to controls, including elevated HR, abbreviated QT intervals, diminished QRS complex amplitudes, prolonged T-wave durations, and ST-segment deviations (*p* < 0.05).

Correlations between Hematological Parameters and Electrocardiographic Abnormalities (Table 3)

Hemoglobin (Hb) and Serum Iron Deficits:

Strong inverse correlations observed between hemoglobin/iron levels and cardiac stress markers:

Elevated heart rate (HR) (ρ = -0.62, *p* < 0.001), indicative of compensatory tachycardia to offset anemia-induced hypoxia. Pronounced ST-segment deviations (ρ = -0.68, *p* < 0.001), *suggesting repolarization abnormalities.* While we excluded structural cardiac pathology via biomarkers (troponin I, NT-proBNP) and echocardiography, detailed functional parameters like ejection fraction (EF), fractional shortening (FS), or diastolic dysfunction markers (E/A ratio, E/E') were not assessed. Future studies should evaluate these to distinguish ischemic vs. repolarization mechanisms.

Packed Cell Volume (PCV) and Ventricular Repolarization

Reduced Hct demonstrated a moderate negative correlation with abbreviated QT intervals (ρ = 0.54, *p* = 0.003), potentially reflecting accelerated ventricular repolarization due to altered ion flux in hypoxic cardiomyocytes. The observed hypoxia-driven changes not directly quantified via blood gases or oximetry. Incorporating these metrics in future, work would strengthen hypoxia severity assessment [41].

Ferritin Depletion and T-Wave Prolongation:

Diminished ferritin levels correlated with prolonged T-wave durations (ρ = -0.58, *p* = 0.001), a finding consistent with iron-dependent disruptions in potassium rectifier currents (IKr) and delayed repolarization [6, 9].

Predicting Anemia Severity (Table 4)

Dogs with severe iron deficiency anemia (Hb <7 g/dL) demonstrated significant hemodynamic and electrophysiological derangements compared to moderately anemic counterparts (Hb ≥7 g/dL):

Elevated heart rate (168.4 ± 32.1 bpm vs. 132 ± 29.5 bpm; *p* < 0.01), consistent with compensatory tachycardia to mitigate systemic hypoxia.

Pronounced ST-segment deviations (0.31 ± 0.12 mV vs. 0.15 ± 0.1 mV; *p* < 0.01), align with repolarization abnormalities rather than ischemia, given the exclusion of dogs with elevated troponin I/NT-proBNP. This supports hypoxia-induced ion channel dysregulation (e.g., K^+ /Ca^{2+} currents) as the primary mechanism, consistent with Chung et al. (2019) [31].

Reduced serum iron (63.2 ± 18.4 µg/dL vs. 97.8 ± 25.1 µg/dL; *p* < 0.01), reflecting progressive iron depletion and impaired oxygen-carrying capacity.

Discussion

The findings from this study align with and expand upon existing research on anemia-related hematological and cardiovascular changes in dogs. Key results demonstrate that anemia profoundly impacts both laboratory indices and ECG parameters, with iron deficiency playing a central role in driving these abnormalities.

Hematological and Iron Profile Abnormalities

The hematological and iron profile findings in this study underscore the profound systemic impact of IDA in dogs, aligning with established pathophysiological mechanisms while revealing species-specific nuances. Marked reductions in erythrocyte count (RBCs: 4.54 ± 0.13 ×10^6 /µL vs. 6.79 ± 0.10 ×10^6 /µL; *p* < 0.001), Hb: 7.71 ± 0.22 g/dL vs. 17.73 ± 0.33 g/dL; *p* < 0.001, and packed cell volume (PCV; 19.01 ± 0.58% vs. 46.52 ± 0.82%; *p* < 0.001) reflected the hallmark triad of IDA impaired erythropoiesis, hemoglobin synthesis, and oxygen transport [1,30]. The microcytic (MCV: 40.23 ± 1.45 fL vs. 67.78 ± 1.03 fL; *p* < 0.001) and hypochromic (MCH: 16.35 ± 0.52 pg vs. 25.92 ± 0.44 pg; *p* < 0.001) erythrocyte morphology further corroborate chronic iron scarcity, as iron is essential for heme synthesis and erythroblast maturation [44].

The paradoxical elevation in MCHC (41.85 ± 0.88 g/dL vs. 38.20 ± 0.69 g/dL; *p* = 0.001) may reflect cellular dehydration secondary to iron-deficient erythropoiesis, a phenomenon previously observed in chronic anemia states [31]. This contrasts with human IDA, where MCHC typically declines, highlighting species-specific adaptations to iron depletion [4].

The iron profile findings—depleted serum iron (90.34 ± 10.23 µg/dL vs. 155.21 ± 8.92 µg/dL; *p* < 0.001), ferritin (75.21 ± 8.45 ng/mL vs. 168.33 ± 12.34 ng/mL; *p* < 0.001), and TIBC (245.67 ± 18.34 µg/dL vs. 337.45 ± 10.21 µg/dL; *p* < 0.001)—confirm iron-restricted erythropoiesis. The reduced TIBC in canine IDA highlights a fundamental difference in iron metabolism between dogs and humans, likely influenced by inflammation and chronic disease processes. The marginal elevation in UIBC suggests some compensatory regulation of iron-binding proteins, but the overall response appears blunted compared to humans. Incorporating inflammatory markers in future research will enhance our understanding of the interplay between iron metabolism and inflammation in canine anemia. [44].

Thrombocytopenia (4.41 ± 0.20 ×10^3/µL vs. 5.51 ± 0.15 ×10^3/µL; *p* = 0.003) and leukopenia (4.63 ± 0.18 ×10^3/µL vs. 5.89 ± 0.14 ×10^3/µL; *p* < 0.001) in anemic dogs suggested iron's broader role in hematopoiesis. Iron deficiency may impair megakaryocyte and leukocyte progenitor differentiation, a mechanism documented in chronic inflammatory states [16, 18].

ECG Abnormalities and Cardiac Stress

Anemic dogs exhibited physiological adaptations like elevated heart rates (145.6 ± 12.3 bpm vs. 125.4 ± 4.2 bpm; *p* = 0.013), a compensatory mechanism to improve oxygen delivery, and abbreviated QT intervals in anemic dogs (201.5 ± 8.3 ms vs. 223.2 ± 5.1 ms; *p* = 0.022), which suggest accelerated ventricular repolarization due to systemic hypoxia and potential myocardial stress. Though electrolyte imbalances were exclusionary, subclinical ion flux alterations cannot ruled out. These findings align with research indicating that iron deficiency impacts calcium-potassium flux in cardiomyocytes, as demonstrated by Chung et al. (2019), who reported that iron-deficiency anemia reduces cardiac contraction by downregulating RyR2 channels and suppressing SERCA pump activity, leading to impaired calcium cycling and myocardial function [31].

Pronounced ST-segment deviations (0.17 ± 0.02 mV vs. 0.10 ± 0.01 mV; *p* = 0.001) and prolonged T-wave amplitudes (0.64 ± 0.12 mV vs. 0.27 ± 0.08 mV; *p* < 0.001) suggest disrupted ventricular repolarization, likely stemming from iron-dependent mitochondrial dysfunction and impaired ion channel regulation [6, 31]. Such electrophysiological disturbances are exacerbated in chronic anemia, where myocardial iron stores are depleted and compromising cellular energetics [13, 23].

Notably, the attenuated QRS complex amplitude in anemic dogs (1.1 ± 0.1 mV vs. 1.3 ± 0.1 mV; *p* < 0.001) contrasts with human studies, where anemia often correlates with ventricular hypertrophy and increased QRS voltage [8, 32]. In dogs, this finding may reflect myocardial hypoxia-induced depolarization deficits or hypokalemia, both of which disrupt sodium channel kinetics [31]. This interspecies discrepancy underscores the need for veterinary-specific diagnostic criteria, as canine cardiac adaptations to anemia diverge from human pathophysiological models [24].

Correlations between ECG and Laboratory Parameters

The significant negative association between hemoglobin (Hb) levels and heart rate (HR) (Spearman's $\rho = -0.62$, *p* < 0.001) emphasizes the systemic hypoxia induced by anemia as a primary driver of compensatory tachycardia. This aligns with prior research in both human and veterinary medicine, where diminished oxygen delivery triggers sympathetic activation to augment cardiac output [2, 18]. Similarly, the robust inverse correlation between ST-segment deviations and serum iron ($\rho = -0.68$, *p* < 0.001) underscores the critical role of iron in myocardial integrity. Iron deficiency disrupts mitochondrial electron transport chain function, exacerbating oxidative stress and impairing ATP synthesis, which may destabilize ventricular repolarization [21, 23]. These observations are corroborated by Ware et. al, (2021) identified iron-mediated dysregulation of calcium handling proteins (e.g., SERCA2a) as a key contributor to arrhythmogenesis in anemic states [18].

Severe vs. Moderate Anemia

Pronounced disparities in heart rate (HR: 168.4 ± 32.1 bpm vs. 132.6 ± 29.5 bpm; *p* = 0.001) and ST-segment deviations (0.31 ± 0.12 mV vs. 0.15 ± 0.1 mV; *p* = 0.003) between severe (Hb <7 g/dL) and moderate anemia cohorts underscore a gradient of cardiac compromise proportional to anemia severity. This dose-dependent relationship mirrors findings in human medicine, where severe anemia exacerbates cardiovascular strain, elevating risks of heart failure and arrhythmias due to chronic hypoxia and volume overload [32, 45]. In dogs, the exaggerated tachycardia and repolarization abnormalities in severe anemia likely reflect heightened sympathetic activation to counterbalance critically reduced oxygen delivery, compounded by iron-deficient mitochondrial dysfunction [31, 45].

Progressive ST-segment deviation in dogs suffering from severe anemia is a notable electrocardiographic finding that often indicates subendocardial ischemia. This condition arises due to an imbalance between the oxygen supply to the myocardium and its demand, frequently linked to reduced coronary perfusion pressure. Elevated cardiac troponin I (cTnI) levels are typically associated with myocardial injury; however, studies that exclude cases with cTnI levels exceeding 0.5 ng/mL suggest that the observed ST-segment changes may primarily reflect disturbances in myocardial repolarization rather than direct myocardial damage. This highlights the importance of understanding the underlying mechanisms and utilizing ECG monitoring as a practical tool for assessing the severity of anemia, particularly in clinical settings where advanced diagnostic imaging may not be available [45-46]. Furthermore, the stark HR differences highlight the clinical utility of ECG monitoring to stratify anemia severity, particularly in settings lacking advanced imaging [45].

Conclusion

Dogs with iron deficiency anemia demonstrate distinct cardiac abnormalities on ECG compared to healthy individuals. These include faster heart rates, diminished electrical signal strength during ventricular activation, shortened recovery phases between heartbeats, prolonged repolarization waves, and irregular shifts in baseline cardiac electrical activity. Such patterns arise from the body's attempt to compensate for reduced oxygen availability through increased cardiac effort, combined with oxygen deprivation in heart tissue, insufficient iron stores, and disruptions in mineral balance. Accelerated electrical conduction between heart

chambers may reflect heightened nervous system stimulation. While parallels exist with anemia-related cardiac stress observed across species, dogs exhibit unique cardiac responses, such as weaker ventricular activation signals, contrasting with patterns seen in other mammals. Timely recognition of these changes is essential to prevent worsening heart complications. Proactive management including dietary correction, targeted iron restoration, and vigilant monitoring of heart rhythm should be prioritized to safeguard cardiovascular health in affected dogs.

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Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

Ethical of approval

This study was conducted in accordance with the ethical standards for clinical research mandated by the Department of Internal Medicine, Faculty of Veterinary Medicine, Cairo University, and aligned with the ARRIVE 2.0 guidelines for rigorous reporting in preclinical animal studies.

TABLE 1. Statistical analysis of Hematological and iron profile

Parameter	Normal Group (Mean ± SE, SD)	Anemic Group (Mean ± SE, SD)	p-value
RBCs (10^6/µL)	6.79 ± 0.10, 0.63	4.54 ± 0.13, 0.84	<0.001
Hb (g/dL)	17.73 ± 0.33, 2.09	7.71 ± 0.22, 1.42	<0.001
PCV (%)	46.52 ± 0.82, 5.21	19.01 ± 0.58, 3.71	<0.001
MCV (fL)	67.78 ± 1.03, 6.54	40.23 ± 1.45, 9.29	<0.001
MCH (pg)	25.92 ± 0.44, 2.82	16.35 ± 0.52, 3.33	<0.001
MCHC (g/dL)	38.20 ± 0.69, 4.41	41.85 ± 0.88, 5.64	0.001
Platelets (10^3/µL)	5.51 ± 0.15, 0.95 (n=15)	4.41 ± 0.20, 1.05 (n=15)	0.003
WBCs (10^3/µL)	5.89 ± 0.14, 0.90 (n=15)	4.63 ± 0.18, 1.01 (n=15)	<0.001
Serum Iron (µg/dL)	155.21 ± 8.92, 56.42	90.34 ± 10.23, 64.85	<0.001
UIBC (µg/dL)	236.15 ± 9.87, 62.46	242.45 ± 15.21, 96.34	<0.001
Ferritin (ng/mL)	168.33 ± 12.34, 78.12	75.21 ± 8.45, 53.67	<0.001
TIBC (µg/dL)	337.45 ± 10.21, 64.58	245.67 ± 18.34, 116.21	<0.001

Normal (n= 60) vs. Anemic (n = 40) Canines (Mean ± SE, SE).

TABLE 2. Statistical analysis of ECG parameters

Parameter	Normal Group (Mean ± SE, SD)	Anemic Group (Mean ± SE, SD)	p-value
HR (bpm)	125.4 ± 4.2, 33.7	145.6 ± 7.8, 38.2	0.013
P Wave Length (mV)	0.16 ± 0.01, 0.08	0.18 ± 0.02, 0.09	0.284
PR Interval (ms)	108.5 ± 3.5, 25.7	94.2 ± 6.1, 27.9	0.048
QRS Amplitude (mV)	1.3 ± 0.1, 0.7	1.1 ± 0.1, 0.5	0.013
QT Interval (ms)	223.2 ± 5.1, 29.8	201.5 ± 8.3, 36.1	0.022
T Wave Length (mV)	0.27 ± 0.02, 0.13	0.64 ± 0.06, 0.25	<0.001
ST segment deviations (mV)	0.10 ± 0.01, 0.06	0.17 ± 0.02, 0.08	0.001

Normal (n= 60) vs. Anemic (n = 40) Canines (Mean ± SE, SE).

TABLE 3. Spearman's Correlations Between Laboratory Indices and ECG

Lab Parameter	ECG Parameter	Effect (Mean ± SE, SD)	p-value
Hemoglobin (Hb)	**HR (bpm)**	Low Hb (<8 g/dL): 158.2 ± 6.2, 24.1 High Hb (≥8 g/dL): 132.4 ± 5.1, 18.2	<0.001
Serum Iron	**ST deviations (mV)**	Low Iron: 0.21 ± 0.03, 0.10 High Iron: 0.12 ± 0.02, 0.07	<0.001
PCV (%)	**QT Interval (ms)**	Low PCV (<20%): 192.3 ± 8.5, 32.4 High PCV (≥20%): 225.4 ± 8.1, 29.7	0.003
Ferritin	**T Wave Length (mV)**	Low Ferritin: 0.81 ± 0.07, 0.28 High Ferritin: 0.52 ± 0.05, 0.20	0.001
MCV	**PR Interval (ms)**	Low MCV: 88.3 ± 5.2, 18.3 High MCV: 102.1 ± 4.8, 16.5	0.021
ECG Parameter	**Lab Parameter**	**P**	**p-value**
HR	**Hb**	-0.62	<0.001*
QRS length	**PCV**	-0.51	0.003*
ST deviations	**Serum Iron**	-0.68	<0.001*
PR interval	Ferritin	-0.45	0.01*

(Mean ± SE, SD)

Table 4. Linear Regression Model Predicting Hemoglobin Levels

Parameter	Severe	Moderate	p-value
HR (bpm)	168.4 ± 32.1	132.6 ± 29.5	0.001*
ST deviations / depression (mV)	0.31 ± 0.12	0.15 ± 0.10	0.003*
Serum Iron	63.2 ± 18.4	97.8 ± 25.1	0.002*

Severe Anemia (Hb < 7 g/dL, n=18) vs. Moderate Anemia (Hb ≥ 7 g/dL, n=22) (Mean ± SD)

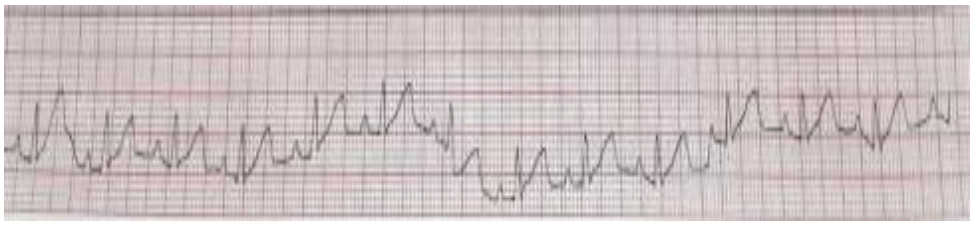

Fig. 1. Electrocardiogram (lead II, sensitivity 1, paper speed 50 mm/s) of a 2-year-old male Mongrel dog suffering from IDA showing short R wave (''R'' 0.7mV), ST segment elevation (0.2mV) and tall T wave (0.5mV)

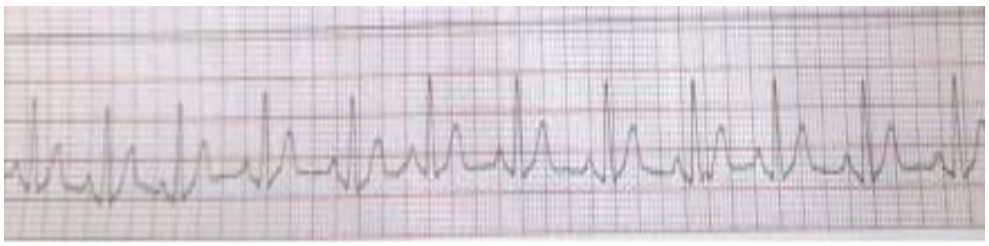

Fig. 2. Electrocardiogram (lead II, sensitivity 1, paper speed 50 mm/s) of a 1-year-old female bitball dog suffering from IDA showing ST segment elevation (0.3 mV), tall T wave (0.5 mV)

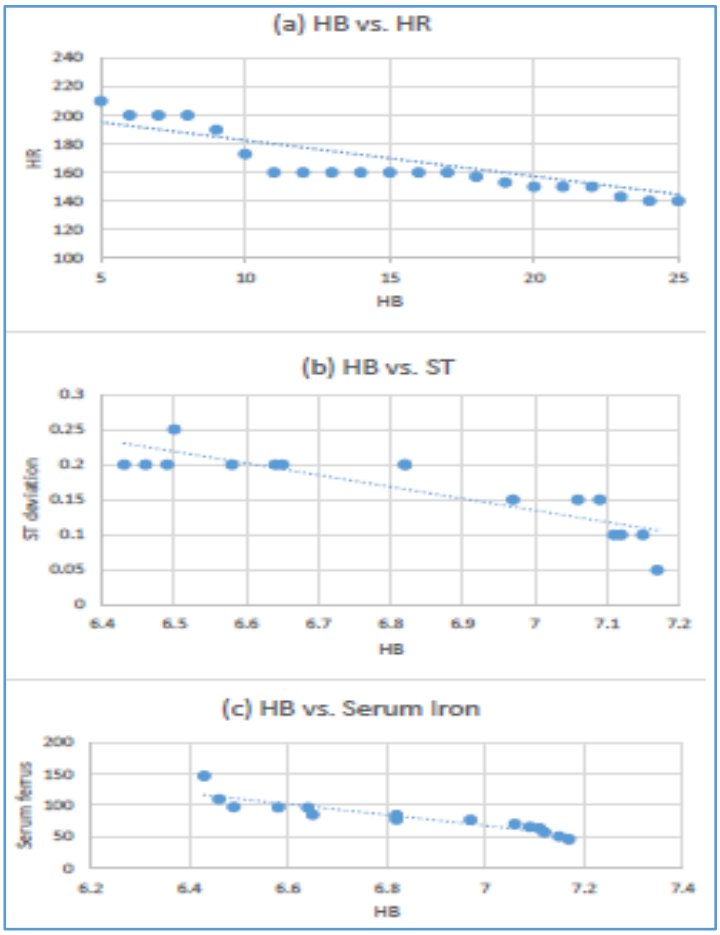

Fig. 3. Linear Regression Model Predicting Hemoglobin Levels verse a(HR), b(ST deviation), and c(serum iron) .

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تحليل إجهاد عضلة القلب: التغيرات في تخطيط كهربية القلب والمؤشرات الدموية في حالات فقر الدم الناجم عن نقص الحديد لدى الكلاب

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الملخص

فقر الدم الناجم عن نقص الحديد، وهو حالة شائعة لكنها غالبًا ما تُهمَل في الكلاب، يُهدد الصحة العامة ويشمل اضطرابات في وظائف القلب والأوعية الدموية. بينما سُجلت تغيرات في تخطيط القلب المرتبطة بفقر الدم لدى البشر والقطط، لا تزال الأدلة حول الكلاب محدودة. اعتمدت هذه الدراسة تصميمًا بحثيًّا يقارن بين الحالات المرضية والمجموعة الضابطة لتقييم التغيرات في تخطيط القلب لدى الكلاب المصابة بفقر الدم الناجم عن نقص الحديد وارتباطها بشدة الأعراض الدموية. شملت العينة مجموعة من الكلاب المُصابة والمؤكدة تشخيصيًا عبر فحوصات دموية محددة، ومجموعة ضابطة من الكلاب السليمة مُطابقة في العمر. خضعت جميع الحيوانات لفحوصات دموية قياسية وتقييم بتخطيط القلب متعدد المسارات، مع تحليل معايير مثل معدل ضربات القلب، وسعة المركبات القلبية، وشكل قطعة التخطيط، والفاصل الزمني للاستقطاب.أظهرت النتائج وجود اختلافات واضحة في تخطيط القلب بين الكلاب المُصابة والضابطة، تشمل ارتفاعًا ملحوظًا في معدل ضربات القلب، وانخفاضًا في سعة المركبات الكهربية، وتقصيرًا في فترات الاستقطاب، وانحرافات في قطعة التخطيط. كما كشفت التحاليل الدموية عن علاقة عكسية بين مستويات الهيموجلوبين ومعدل ضربات القلب، وبين تركيز الحديد في الدم وشدة الانحرافات في التخطيط. عند تصنيف الحالات وفقًا لشدة فقر الدم، لوحظ تفاقم أكبر في تسارع ضربات القلب وشذوذات التخطيط في الحالات المتقدمة.تشير هذه النتائج إلى أن فقر الدم الناجم عن نقص الحديد يُسبب نقصًا في أكسجة عضلة القلب واختلالًا في التوازن الأيوني، مما يؤدي إلى اضطرابات كهربية تظهر كتغيرات في استقطاب القلب وزيادة تعويضية في معدل ضرباته. يختلف نمط التغيرات في التخطيط عن الملاحظ في البشر، مما يُؤكد على خصوصية التكيفات القلبية لدى الكلاب. تُسلط الدراسة الضوء على أهمية استخدام تخطيط القلب في الكشف المبكر عن المضاعفات القلبية المرتبطة بفقر الدم، خاصة في الظروف محدودة الإمكانيات. هناك حاجة لدراسات إضافية لتحديد معايير مرجعية خاصة بكل سلالة، وتقييم تحسن التخطيط بعد العلاج. تساهم هذه النتائج في سد فجوة معرفية في طب القلب البيطري، وتُقدم أساسًا لتحسين التدخلات العلاجية للحد من الاعتلالات القلبية المرتبطة بالأنيميا.

الكلمات الدالة: فقر الدم الناجم عن نقص الحديد في الكلاب, تخطيط كهربية القلب, نقص أكسجة عضلة القلب, الارتباط الدموي, اختلال وظائف القلب والأوعية الدموية.