

International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
IJABR 2025; SP-9(1): 281-290
www.biochemjournal.com
Received: 02-11-2024
Accepted: 05-12-2024

Abhishek Dhiman
Department of Veterinary
Medicine, Dr. GC Negi College of
Veterinary and Animal Sciences
CSK Himachal Pradesh Krishi
Vishwavidyalaya Palampur,
Himachal Pradesh, India

Pardeep Sharma
Department of Veterinary
Medicine, Dr. GC Negi College of
Veterinary and Animal Sciences
CSK Himachal Pradesh Krishi
Vishwavidyalaya Palampur,
Himachal Pradesh, India

Deepti Bodh
Department of Veterinary
Surgery & Radiology, Dr. G.C.
Negi College of Veterinary and
Animal Sciences, CSK HP Krishi
Vishwavidyalaya, Kangra,
Himachal Pradesh, India

Ajay Katoch
Department of Veterinary
Medicine, Dr. G.C. Negi College
of Veterinary and Animal
Sciences, CSK HP Krishi
Vishwavidyalaya, Kangra,
Himachal Pradesh, India

Ankur Sharma
Department of Veterinary
Medicine, Dr. G.C. Negi College
of Veterinary and Animal
Sciences, CSK HP Krishi
Vishwavidyalaya, Kangra,
Himachal Pradesh, India

Adarsh Kumar
Department of Veterinary
Surgery & Radiology, Dr. G.C.
Negi College of Veterinary and
Animal Sciences, CSK HP Krishi
Vishwavidyalaya, Kangra,
Himachal Pradesh, India

Corresponding Author:
Pardeep Sharma
Department of Veterinary
Medicine, Dr. GC Negi College of
Veterinary and Animal Sciences
CSK Himachal Pradesh Krishi
Vishwavidyalaya Palampur,
Himachal Pradesh, India

Evaluating treatment protocols for congestive heart failure in geriatric dogs

Abhishek Dhiman, Pardeep Sharma, Deepti Bodh, Ajay Katoch, Ankur Sharma and Adarsh Kumar

DOI: <https://doi.org/10.33545/26174693.2025.v9.i1Sd.3481>

Abstract

This study evaluated the efficacy of different therapeutic protocols in managing congestive heart failure (CHF) in geriatric dogs. Twenty-four dogs diagnosed with CHF were randomly categorized into three groups: Group I (digoxin), Group II (pimobendane), and Group III (combination of carvedilol and pimobendane). Haematological and biochemical parameters were assessed at baseline (day 0) and post-treatment (day 42). Clinical outcomes and cardiac biomarkers (cardiac troponin-I), and survival rates were also analyzed at day 0 and day 42. In conclusion, Group III demonstrated the most significant improvements, including normalization of haematological and biochemical parameters, a significant reduction in cardiac troponin-I levels, and the highest survival rate (66.67%). These results indicated the superiority of the combined therapy, especially in cases with severe tachycardia, hypertension, and atrial fibrillation.

Keywords: Carvedilol, digoxin, geriatric dogs, survival

Introduction

The incidence of heart disease increases dramatically with age. In canines, heart diseases can occur at any age, but the risk increases significantly in older dogs who have completed 75–80% of their lifespan, with age being the primary risk factor for cardiac issues (Urfer *et al.*, 2017) [23]. Cardiac diseases can be acquired or congenital, with the former accounting for 95% of all cardiac conditions. These conditions can be classified based on their source into valvular, myocardial, and pericardial diseases (Hoque *et al.*, 2019) [9]. The common ultimate pathological feature for all cardiovascular diseases is congestive heart failure (CHF). Congestive heart failure can arise as a severe complication of various cardiac disorders (Beaumier *et al.*, 2018) [4]. The management and treatment for dogs with CHF usually involves the administration of diuretics, positive inotropic drugs, oxygen supplementation, anti-hypertensive drugs, cardiac diets and other medications. Effusions may be manually drained through procedures like abdominocentesis, thoracocentesis, or pericardiocentesis. In recent times, the utilization of pimobendane alongside or as a substitute for an angiotensin-converting enzyme inhibitor has been found to improve symptoms and quality of life (Haggstrom *et al.*, 2008) [8]. Although medications or other procedures can enhance the quality of life and survival rates in patients with congestive heart failure, they do not address the underlying heart disease that causes CHF. Hence, once a dog develops congestive heart failure, the chances of achieving full recovery, even with treatment, are very slim (Beaumier *et al.*, 2018) [4]. Considering these critical insights, the present study was designed with the objective of evaluating therapeutic strategies for managing congestive heart failure in geriatric dogs, aiming to optimize treatment outcomes and improve their quality of life.

Materials and Methods

The present study was conducted *w.e.f.* June 01, 2023, to August 31, 2024, in the Department of Veterinary Medicine, Dr. G. C. Negi College of Veterinary & Animal Sciences, CSKHPKV Palampur, Himachal Pradesh. During the study period, the number of dogs brought to the Department of Veterinary Medicine was 2,574. Of these, 275 geriatric canines were screened and 24 were diagnosed with congestive heart failure (CHF) and the diagnosed cases were randomly subjected to the therapeutic trials as shown in Table 1.

Table 1: Therapeutic groups for the treatment of CHF affected geriatric dogs

Sr. No.	Groups	DRUGS			
		Diuretics	Positive Inotropes	ACE Inhibitor	Anti-arrhythmic drugs
1.	I (n=6)	Furosemide @ 2 mg/kg BW + Spironolactone @ 2 mg/kg BW, twice daily orally	Digoxin @ 0.005 mg/kg of body weight (BW), twice daily orally	Enalapril / Benazepril @ 0.25 – 0.5 mg/kg BW, twice daily orally	-----
2.	II (n=9)	Furosemide @ 2 mg/kg BW + Spironolactone @ 2 mg/kg BW, twice daily orally	Pimobendane @ 0.25 – 0.5 mg/kg BW, twice daily orally	Enalapril / Benazepril @ 0.25 – 0.5 mg/kg BW, twice daily orally	-----
3.	III (n=6)	Furosemide @ 2 mg/kg BW+ Spironolactone @ 2 mg/kg BW, twice daily orally	Pimobendane @ 0.25 – 0.5 mg/kg BW, twice daily orally	Enalapril / Benazepril @ 0.25 – 0.5 mg/kg BW, twice daily orally	Carvedilol @ 0.1-0.2 mg/kg BW, twice daily orally

The choice of treatment was influenced by the owner's willingness and financial capacity to invest in their pet's care, as pimobendane was comparatively more expensive than digoxin. Also, Group III treatment was specifically prioritized for dogs presenting with severe tachycardia (>200 bpm), atrial fibrillation, and/or significant hypertension (>190 mmHg), ensuring optimal care for critical cases.

Supportive management includes

Thoracocentesis (chest tap) and abdominocentesis (fluid removal from the abdomen) were performed for diagnostic or therapeutic purposes in cases of fluid or air accumulation. Medications such as butorphanol (0.2 mg/kg IV/IM SOS), diltiazem (0.5-1 mg/kg PO BID), and sildenafil (2 mg/kg PO BID) were strategically utilized based on individual patient needs. Hyperkalemia was managed with furosemide (2-4 mg/kg for 2 days) followed by maintenance with furosemide (2 mg/kg) and spironolactone (2 mg/kg) BID. Antibiotics like doxycycline and ceftriaxone were used as needed. Owners were advised to limit physical activity, avoid high-sodium diets, and provide high-protein food (e.g., egg whites, homemade, or commercial cardiac diets). Supplements like taurine, Vitamin E, CoQ10, folic acid, selenium, and L-carnitine were also given.

Table 2: Various nutraceutical drugs used for management of CHF along with trial drugs.

Sr. No.	Product Name	Dosage
1.	Tab. Strongbeat (By Savavet Manufacturers)	@ 1 tab. / 16 Kg of BW
2.	Tab. Cardio Support (By Vetina Manufacturers)	@ 1 tab. / 10 kg of BW
3.	Tab. Tauritine Plus (By Oriheal Manufacturers)	@ 1 tab. / 10 kg of BW

Follow-up evaluations were conducted on day 15 and day 42 of post-treatment. Each dog underwent a minimum of two examinations, with most receiving three. However, for the purpose of our study, only data from day 0 (baseline) and day 42 (final assessment) were analysed and included. The different treatment regimens were evaluated at day zero and 42nd based on history, clinical signs, haematological, biochemical, electrolytes, electrocardiography findings and various other parameters. To assess the effectiveness of therapy, eight indicators were chosen: ascites, anorexia, nocturnal coughing, dyspnoea, syncope, retching, arrhythmia and exercise intolerance. Each variable was scored based on the patient's progress: 0 for complete resolution of symptoms, 1 for noticeable improvement without full recovery, 2 for no change, and 3 for worsening symptoms

during treatment. The average score for each variable was calculated to evaluate the overall response to therapy, allowing for comparison between different patient groups. The collected data was statistically analysed using Instat software from Graphpad (2008) and SPSS. Mean values of various parameters between control and diseased groups, as well as pre- and post-treatment data, were compared. Statistical significance at different levels was determined using the t-test and ANOVA, providing a thorough evaluation of the treatment effects.

Results and Discussion

In the present study, the occurrence of congestive heart failure in geriatric dogs was recorded at nearly 1% (0.93%). The haematological parameters remained within normal ranges, as shown in Tables 3, 4 and 5. This finding aligns with earlier research by Olsen *et al.*, (2010) [13], who noted that haematology is primarily useful for ruling out other diseases. In Group I, Table 3 all haematological parameters were normal except on day 0 with the total erythrocyte count (TEC) and packed cell volume (PCV) were low, while mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were elevated, this is in contrast to the findings of Tangmahakul *et al.*, (2023) [21], who found decrease in MCH and MCHC in MMVD, MMVD + pulmonary hypertension (PH) and PH groups compared to the normal dogs. The low PCV may indicate anaemia, consistent with the findings of Martin (2009) [11]. The increased MCH could be attributed to the low TEC. In Group II, Table 4 all haematological parameters were normal at day 0, except for elevated total leukocyte count (TLC) and red cell distribution width (RDW). Similarly, in Group III, Table 5 all haematological parameters were normal at day 0 except for low values of TEC, PCV, and haemoglobin, alongside high RDW values. Reece (2004) [15], suggested that decreases in haemoglobin, PCV, and TEC could indicate ischemia, pointing to potential cardiovascular disorders.

The impact of treatment on haematology parameters at day 42, as shown in Tables 3, 4, and 5, revealed mostly non-significant variations between pre- and post-treatment levels. However, notable improvements were observed in specific groups. In Group I, Table 3, the levels of TEC, PCV, MCH, and MCHC all returned to normal ranges. Group II saw normalization in TLC values, although RDW levels remained elevated, as depicted in Table 4. Meanwhile, Group III demonstrated in Table 5, a complete normalization in TEC, haemoglobin, and PCV levels. These findings highlight distinct responses across the groups, emphasizing the differential effects of treatment on various haematology parameters.

Table 3: Haematological changes in group I pre-treatment and post-treatment

Sr. No.	Parameters	Control (n=6)	Day 0 (Pre-Treatment) (n=6)	Day 42 (Post-Treatment) (n=4)
1.	TLC ($\times 10^9/L$)	11.53 $\pm$ 1.02	12.81 $\pm$ 2.11	13.04 $\pm$ 2.10
2.	Neu ($\times 10^9/L$)	9.9 $\pm$ 1.10	11.58 $\pm$ 2.08	8.96 $\pm$ 1.54
3.	Lymphocytes ($\times 10^9/L$)	2.01 $\pm$ 0.40	1.36 $\pm$ 0.20	0.98 $\pm$ 0.19
4.	Monocytes ($\times 10^9/L$)	0.50 $\pm$ 0.11	1.30 $\pm$ 0.80	1.185 $\pm$ 0.09
5.	Eosinophils ($\times 10^9/L$)	0.25 $\pm$ 0.05	0.30 $\pm$ 0.22	0.56 $\pm$ 0.05
6.	TEC ($\times 10^{12}/L$)	6.56 $\pm$ 0.25 ^a	4.85 $\pm$ 0.49 ^b	5.89 $\pm$ 0.63 ^{ab}
7.	Haemoglobin (g/dL)	13.65 $\pm$ 0.70	12.19 $\pm$ 1.61	11.39 $\pm$ 0.82
8.	PCV %	39.77 $\pm$ 0.91 ^a	27.40 $\pm$ 2.30 ^b	34.25 $\pm$ 0.94 ^a
9.	MCV (fL)	58.52 $\pm$ 1.48	62.04 $\pm$ 1.20	64.58 $\pm$ 3.18
10.	MCH (pg)	21.22 $\pm$ 0.52 ^a	23.83 $\pm$ 0.70 ^b	21.83 $\pm$ 0.47 ^{ab}
11.	MCHC	33.34 $\pm$ 0.75 ^a	38.31 $\pm$ 0.49 ^b	33.27 $\pm$ 1.47 ^a
12.	RDW %	13.55 $\pm$ 0.30	18.90 $\pm$ 2.67	12.78 $\pm$ 0.61
13.	Platelets ($\times 10^9/L$)	299.17 $\pm$ 25.50	277.16 $\pm$ 54.62	284.75 $\pm$ 19.28

Mean bearing different superscripts within the same rows differ significantly (p<0.05)

Table 4: Haematological changes in group II pre-treatment and post-treatment

Sr. No.	Parameters	Control (n=6)	Day 0 (Pre-Treatment) (n=9)	Day 42 (Post-Treatment) (n=6)
1.	TLC ($\times 10^9/L$)	11.53 $\pm$ 1.02 ^a	70 $\pm$ 7.018 ^b	11.44 $\pm$ 2.25 ^a
2.	Neu ($\times 10^9/L$)	9.90 $\pm$ 1.10	10.11 $\pm$ 2.2	9 $\pm$ 1.92
3.	Lymphocytes ($\times 10^9/L$)	2.01 $\pm$ 0.40	1.80 $\pm$ 0.30	1.65 $\pm$ 0.31
4.	Monocytes ($\times 10^9/L$)	0.50 $\pm$ 0.11	0.66 $\pm$ 0.13	0.53 $\pm$ 0.10
5.	Eosinophils ($\times 10^9/L$)	0.25 $\pm$ 0.05	0.24 $\pm$ 0.08	0.39 $\pm$ 0.18
6.	TEC ($\times 10^{12}/L$)	6.56 $\pm$ 0.25	5.00 $\pm$ 0.70	5.46 $\pm$ 0.78
7.	Haemoglobin (g/dL)	13.65 $\pm$ 0.70	10.12 $\pm$ 1.5	11.88 $\pm$ 1.59
8.	PCV %	39.77 $\pm$ 0.91	27.95 $\pm$ 4.05	28.30 $\pm$ 4.73
9.	MCV (fL)	58.52 $\pm$ 1.48	63.50 $\pm$ 1.50	60.91 $\pm$ 2.01
10.	MCH (pg)	21.22 $\pm$ 0.52	22.61 $\pm$ 0.65	22.27 $\pm$ 0.70
11.	MCHC	33.34 $\pm$ 0.75	35.70 $\pm$ 0.62	36.16 $\pm$ 1.08
12.	RDW %	13.55 $\pm$ 0.30 ^a	16.14 $\pm$ 0.76 ^b	18.36 $\pm$ 0.85 ^b
13.	Platelets ($\times 10^9/L$)	299.17 $\pm$ 25.50	206.00 $\pm$ 65.70	249.00 $\pm$ 60.34

Mean bearing different superscripts within the same rows differ significantly (p<0.05)

Table 5: Haematological changes in group III pre-treatment and post-treatment

Sr. No.	Parameters	Control (n=6)	Day 0 (Pre-Treatment) (n=6)	Day 42 (Post-Treatment) (n=5)
1.	TLC ($\times 10^9/L$)	11.53 $\pm$ 1.02	10.44 $\pm$ 0.73	10.87 $\pm$ 0.66
2.	Neu ($\times 10^9/L$)	9.90 $\pm$ 1.10	8.08 $\pm$ 0.60	8.6 $\pm$ 1.00
3.	Lymphocytes ($\times 10^9/L$)	2.01 $\pm$ 0.40	1.51 $\pm$ 0.39	1.42 $\pm$ 0.38
4.	Monocytes ($\times 10^9/L$)	0.50 $\pm$ 0.11	0.67 $\pm$ 0.13	0.46 $\pm$ 0.14
5.	Eosinophils ($\times 10^9/L$)	0.25 $\pm$ 0.05	0.175 $\pm$ 0.07	0.186 $\pm$ 0.09
6.	TEC ($\times 10^{12}/L$)	6.56 $\pm$ 0.25 ^a	4.35 $\pm$ 0.40 ^b	5.35 $\pm$ 0.35 ^{ab}
7.	Haemoglobin (g/dL)	13.65 $\pm$ 0.70 ^a	9.11 $\pm$ 1.12 ^b	11.23 $\pm$ 0.42 ^{ab}
8.	PCV %	39.77 $\pm$ 0.91 ^a	24.68 $\pm$ 3.30 ^b	33.19 $\pm$ 3.2 ^{ab}
9.	MCV (fL)	58.52 $\pm$ 1.48	59.85 $\pm$ 5.90	55.92 $\pm$ 6.11
10.	MCH (pg)	21.22 $\pm$ 0.52	22.96 $\pm$ 0.61	21.50 $\pm$ 0.65
11.	MCHC	33.34 $\pm$ 0.75	35.48 $\pm$ 0.98	35.34 $\pm$ 1.22
12.	RDW %	13.55 $\pm$ 0.30 ^a	17.98 $\pm$ 0.90 ^b	17.65 $\pm$ 1.50 ^b
13.	Platelets ($\times 10^9/L$)	299.17 $\pm$ 25.50	173.50 $\pm$ 48.65	218.40 $\pm$ 22.16

Mean bearing different superscripts within the same rows differ significantly (p<0.05)

In our study, the biochemical findings across all diseased groups at day 0 showed no significant differences, in all three groups, when compared to the mean absolute values of the control group animals. This aligns with Ristic (2004) [17], who noted that haematological and biochemical evaluations often lack significant diagnostic value for heart diseases. The exception was Group II, Table 6, where elevated values of alanine aminotransferase (ALT) were observed on day 0. This is consistent with findings from Deepti (2016) [6], and Ristic (2004) [17], who reported that elevated blood urea nitrogen, creatinine, ALT, and aspartate aminotransferase (AST) levels can be attributed to organ congestion due to low cardiac output in dogs with DCM.

The effect of treatment on biochemical parameters for Group I, Group II and Group III, showed largely non-significant changes between pre-and post-treatment levels. However, a noteworthy exception was observed in Group II, Table 6, where ALT values returned to normal by day 42, indicating a positive response to treatment. Despite the general stability across parameters, improvement in ALT was found, may be due to improved cardiac output leads to decrease in the congestion of liver.

In our study, the electrolyte findings for both the control group and those with cardiac diseases at day 0, showed no significant differences in electrolyte levels, when comparing the two groups, which aligns with the results reported by Sharma (2023) [18].

Table 6: Biochemical changes in group II pre-treatment and post-treatment

Sr. No.	Parameters Healthy group	Healthy group (n=6)	Pre-treatment (Day 0) (n=9)	Post-treatment (Day 42) (n=6)
1.	ALT (U/L)	30.45±3.35 ^a	89.17±24.82 ^b	44.11±5.92 ^{ab}
2.	AST (U/L)	29.69±2.23	52.44±8.41	36.89±7.12
3.	ALP (U/L)	64.12±20.10	159.47±62.41	97.11±20.02
4.	Total Bilirubin (mg/dL)	0.13±0.09	0.34±0.09	0.32±0.12
5.	Total Plasma Protein (g/dL)	6.64±0.30	6.60±0.42	6.98±0.60
6.	BUN (mg/dL)	21.55±2.30	116.37±42.32	47.98±9.28
7.	Creatinine (mg/dL)	0.77±0.07	3.25±1.12	1.80±0.11
8.	Blood Glucose (mg/dL)	100.5±10.85	111.49±6.59	108.26±9.31

Mean bearing different superscripts within the same rows differ significantly (p<0.05)

However, other studies, such as those by Thakuria *et al.*, (2018) [21], have noted significant variations in electrolytes, particularly in sodium and potassium levels.

At day 42, the effect of treatment on electrolyte parameters, revealed that all values remained within the normal range across groups. However, in Group III, Table 7, sodium levels were significantly lower (139.18±1.90) mmol/L compared to the control, yet still within the standard range of 135–155 mmol/L. While this slight reduction in sodium is

noteworthy, it falls within acceptable limits, indicating that overall electrolyte balance was maintained despite the treatment. Macdonald *et al.*, (1999) [10] also identified hyponatremia and hypotension as adverse reactions associated with carvedilol treatment. Hyponatremia has been strongly linked to neurohormonal activation in heart failure, particularly involving the renin–angiotensin system, and is recognized as an independent predictor of mortality in patients with severe heart failure (Dzau *et al.*, 1981) [7].

Table 7: Electrolyte changes in group III pre-treatment and post-treatment

Parameters	Healthy group (n=6)	Pre-treatment (Day 0) (n=6)	Post-treatment (Day 42) (n=5)
Sodium (Na ⁺) (mmol/L)	151.05±1.85 ^a	145.88±2.60 ^{ab}	139.18±1.90 ^b
Potassium (K ⁺) (mmol/L)	4.38±0.16	4.47±0.20	4.623±0.310
Chloride (Cl ⁻) (mmol/L)	112.82±1.48 ^a	110.7±0.03 ^{ab}	104.178±3.10 ^b
Ionic Calcium iCa ⁺ (mmol/L)	1.125±0.10	1.305±0.03	1.311±0.04
Total Calcium (tCa ⁺) (mg/dl)	10.325±0.85	10.69±0.14	10.659±0.19

Mean bearing different superscripts within the same rows differ significantly (p<0.05)

Table 8, presents the cardiac biomarker cardiac troponin-I (cTn-I) levels in both the control group and those with cardiac diseases. The cTn-I levels were significantly elevated across all cardiac disease groups. On day 0, Group I, showed a significant increase (p< 0.01). In Group II, cTn-I levels were also significantly elevated (p< 0.01). In Group III, cTn-I was highly significant (p< 0.0001) when compared to the control group. Overall, the cTn-I values were higher in all cardiac disease groups compared to controls, consistent with the findings of Wess *et al.*, (2010) [26], and Ando *et al.*, (2023) [2]. Elevated cTn-I levels occur due to sarcomere injury, which leads to the detachment of cTn-I from actin. This, along with membrane disruption, allows cTn-I to leak into the bloodstream (Reynolds and Oyama, 2008) [16]. The effect of treatment on cardiac troponin I is depicted in Table 8. In Group I, cTn-I levels dropped significantly by day 42. This reduction was statistically significant compared to day 0, but it remained

less pronounced when compared to the reductions observed in other groups. This reduction is due to digoxin primarily works by inhibiting the sodium-potassium ATPase pump in cardiac cells. This inhibition increases intracellular sodium levels, which subsequently boosts intracellular calcium levels via the sodium-calcium exchanger. Increased calcium availability enhances myocardial contractility, thus improving cardiac output and reducing the heart's workload. Lower myocardial stress can reduce the ongoing myocardial injury, which may lead to a reduction in cTn-I levels (Aiello *et al.*, 2016) [1].

In Group II, cTn-I levels saw a highly significant reduction by day 42. By this point, at day 42 the values became non-significant when compared to both the control group and day 0, indicating substantial improvement. Pimobendane's combined vasodilatory and inotropic effects help reduce afterload and enhance cardiac performance (Aiello *et al.*, 2016) [1], thus reducing the levels of cTn-I.

Table 8: Cardiac troponin–I estimation in CHF-affected dogs

Sr. No.	Cardiac Biomarker	Group I		
1.	cTn-I (ng/mL)	Control (n=6)	Pre-treatment (Day 0) (n=4)	Post-treatment (Day 42) (n=4)
		0.1±0.001	1.08±0.29**	0.35±0.10*
2.	cTn-I (ng/mL)	Control (n=6)	Pre-treatment (Day 0) (n=4)	Post-treatment (Day 42) (n=4)
		0.1±0.001	1.12±0.30**	0.13±0.02
3.	cTn-I (ng/mL)	Control (n=6)	Pre-treatment (Day 0) (n=4)	Post-treatment (Day 42) (n=4)
		0.1±0.001	1.35±0.30***	0.25±0.11

*Significant at (p < 0.05) w.r.t. day 0.

**Significant at (p < 0.01) w.r.t. the control group.

***Significant at (p < 0.0001) w.r.t. the control group.

In Group III, cTn-I levels showed a very highly significant reduction by day 42. Like in Group II, the values at day 42 were no longer significant when compared to both the control group and day 0, highlighting a remarkable recovery in cTn-I levels. All these findings are depicted in Table 8. The highest reduction in levels of cTn-I may be because of combining effects of carvedilol and pimobendane. Carvedilol’s antioxidant properties and ability to protect against free radicals may play a role in its positive effects (Avila *et al.*, 2018) [13].

Additionally, carvedilol is a non-selective beta-blocker that slows the heart rate and decreases myocardial oxygen demand by blocking beta-adrenergic receptors. This action reduces sympathetic nervous system activity, lowering cardiac workload and helping to prevent further damage to heart cells (Aiello *et al.*, 2016) [11].

The survivability rate, as shown in Table 9, revealed an overall survival of 41.67% in the study, where fourteen dogs unfortunately passed away while ten survived out of 24 diagnosed cases. This is in contrast to the findings of Beaumier *et al.*, (2018) [4], who found a 31.40% survival rate in dogs with advanced heart failure, and Brloznik (2023) [5] who found a 35.20% of survival rate in dogs affected with mitral valve disease (MVD) and CHF. In our study, the primary causes of death included renal failure, haemo- protozoan diseases, liver-related tumors, severe pneumonia, and jaundice. Additionally, in three cases, the owners declined further treatment.

In Group I, the survival rate was 33.34% (2 dogs), closely matching the results of Tidholm (2006) [22]. Group II had a higher survival rate of 44.45% (4 dogs), indicating a longer survival time compared to Group I. This finding is consistent with Mizuno *et al.*, (2017) [12], who reported prolonged survival times with standard pimobendane therapy, achieving a 46% survival rate.

Group III showed the highest survival rate at 66.67%, with 5 out of 6 dogs surviving. This result mirrors the findings of Soares *et al.*, (2010) [19], who demonstrated that dogs treated with carvedilol alongside conventional therapy had a 45% longer survival time than those without the drug. Soares *et al.*, (2010) [19] also observed that carvedilol contributed to stability in the left ventricular end-systolic diameter indexed to body surface area (LVESDi), suggesting a beneficial effect on ventricular remodelling. As chronic stress on the heart, especially from high sympathetic nervous system activity, often leads to ventricular remodeling—a process where the heart’s structure changes in response to increased demand. Carvedilol blocks this sympathetic stimulation, reducing the risk of adverse remodeling, and helping stabilize LVESDi. Maintaining stable LVESDi prevents the ventricles from becoming excessively dilated. Furthermore, Palazzuolli *et al.*, (2005) [14], in a study on human patients with dilated cardiomyopathy (DCM), noted improved ejection fractions and reduced left ventricular end-systolic volume after a year of carvedilol therapy. These outcomes underscore the potential therapeutic advantages of combining carvedilol with conventional treatment.

Table 9: Survivability rate (post-treatment) in CHF affected geriatric dogs

Sr. No.	Parameter	Group I (n=6)	Group II (n=9)	Group III (n=6)	Overall (n=24)
1.	Survival Rate	2 (33.34%)	4 (44.45%)	4 (66.67%)	10 (41.67%)

Mean scores for resolution of clinical signs and arrhythmias at day 42 in CHF affected geriatric dogs

The scores for the resolution of clinical signs across the four treatment groups, as presented in Table 10, highlight distinct differences in treatment outcomes. Group I recorded the highest mean score of 0.90, indicating slower clinical improvement. Group II followed with a lower score of 0.40, reflecting better resolution of symptoms. Group III, despite experiencing a mortality, achieved the lowest score of 0.25, suggesting the most significant improvement in clinical signs. These results emphasize that Group III demonstrated the best response to treatment, while Group I showed the slowest recovery.

Figures (1-14), showcased the electrocardiographic (ECG) changes before and after treatment in dogs with various cardiac conditions. Figure 1 depicted atrial fibrillation in a French Mastiff with DCM, while Figure 2 highlighted a normalized ECG post-Group III therapy. In a Rottweiler

with MVD, Figure 3 illustrated sinus bradycardia, and Figure 4 demonstrated a restored heart rate after Group II treatment. Similar improvement patterns were observed in other figures, highlighting the effectiveness of tailored therapies in managing ECG abnormalities.

Table 10: Resolution of clinical signs and symptoms in CHF survived cases

Sr. No.	Parameter	Group I	Group II	Group III
1.	Syncope	1.5	1	1
2.	Retching	0.5	0.5	0.5
3.	Nocturnal coughing	1	0.5	0.5
4.	Exercise intolerance	0.5	0	0
5.	Anorexia	1	0	0
6.	Dyspnoea	0.75	0.25	0
7.	Ascites	0.5	0	0
8.	Pathological arrhythmias	1.5	1	0
9.	Total mean	0.90	0.40	0.25

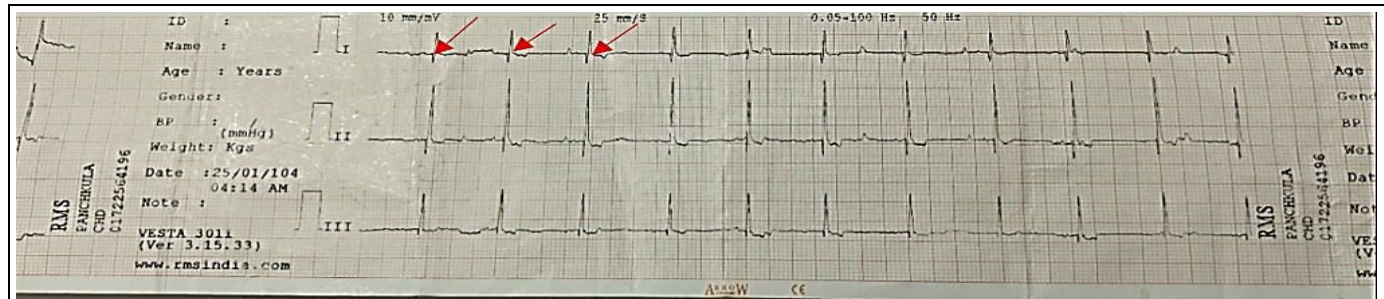


Fig 1: Pre-treatment Atrial fibrillation in a French mastiff diagnosed with DCM (50 mm/s paper speed; 10 mm/mV gain).

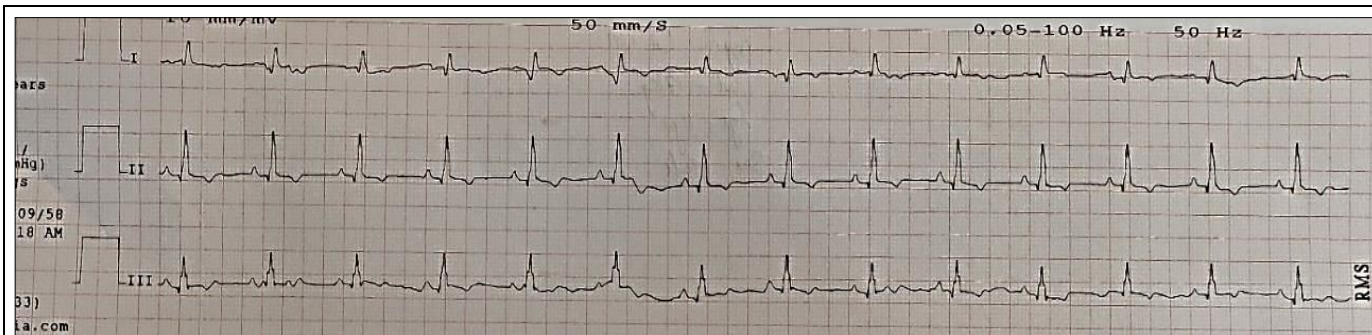


Fig 2: Improvement in ECG of French mastiff diagnosed with DCM after treatment with group III (50 mm/s paper speed; 10 mm/mV gain).

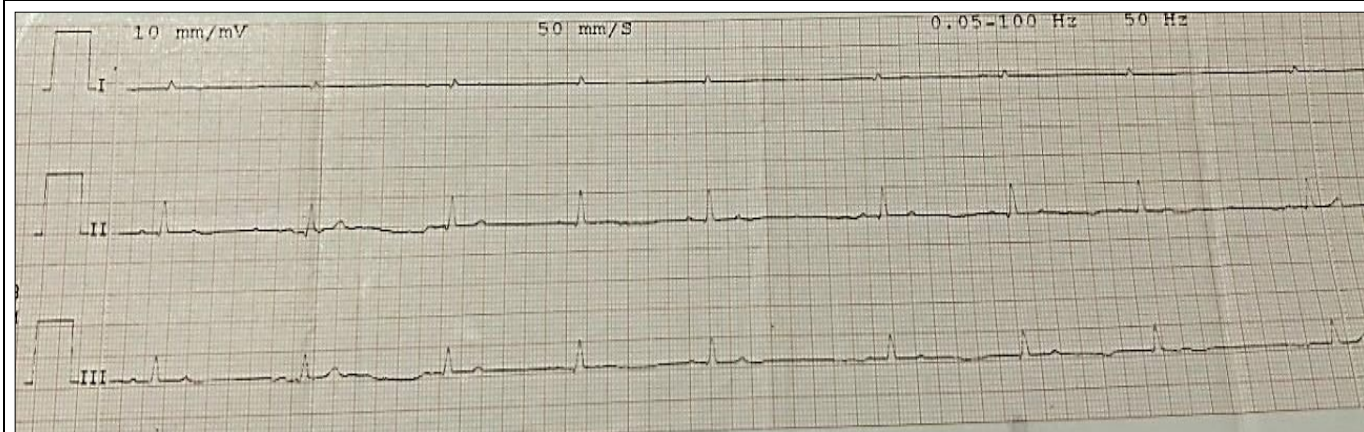


Fig 3: Pre-treatment ECG showing sinus bradycardia (HR < 70 b/m) in a Rottweiler diagnosed with MVD (50 mm/s paper speed; 10 mm/mV gain).

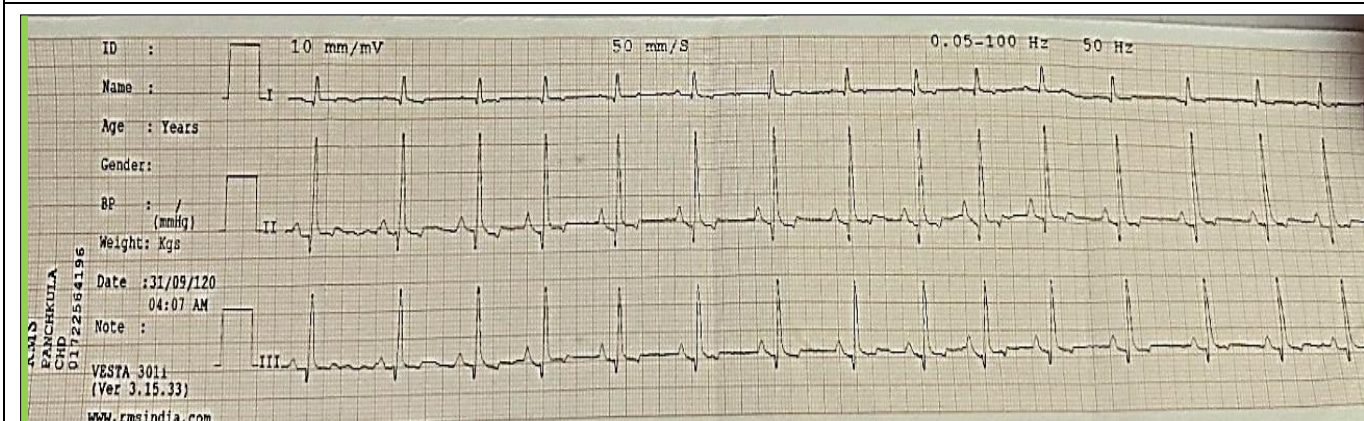


Fig 4: Normal heart rate (HR 110 b/m) in a Rottweiler diagnosed with MVD after treatment with group II (50 mm/s paper speed; 10 mm/mV gain).

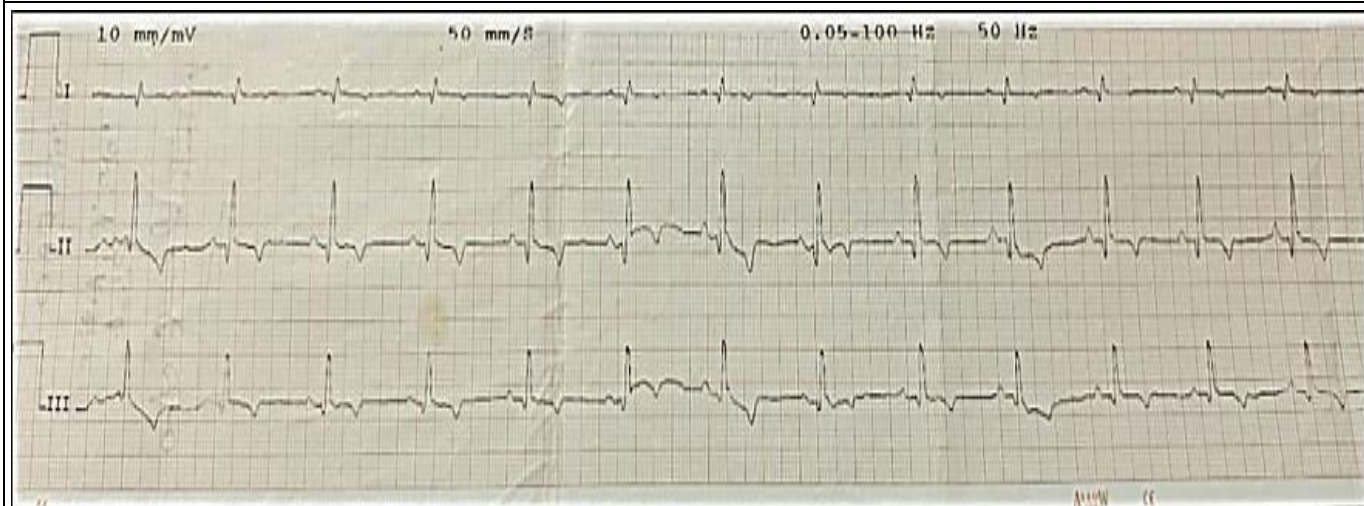


Fig 5: ST elevation, in a dog with hypercalcemia (50 mm/s paper speed; 10 mm/mV gain).

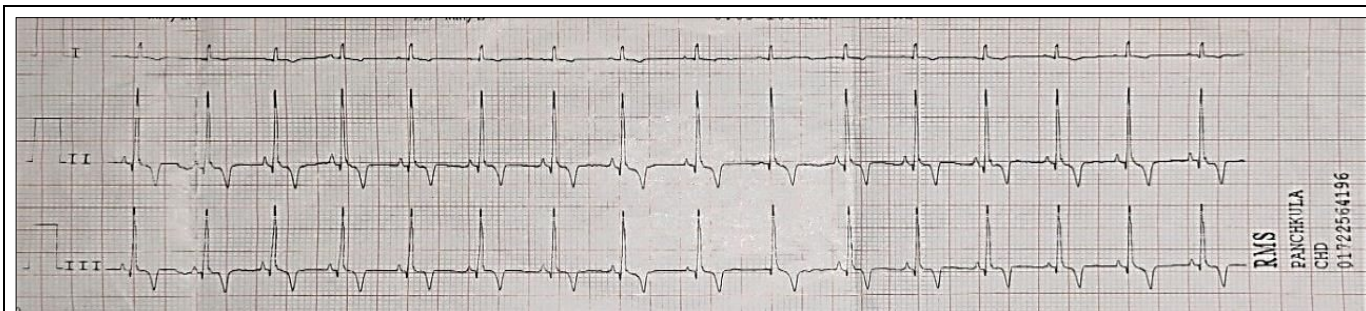


Fig 6: Normal ST elevation after treatment with group III (25 mm/s paper speed; 10 mm/mV gain).

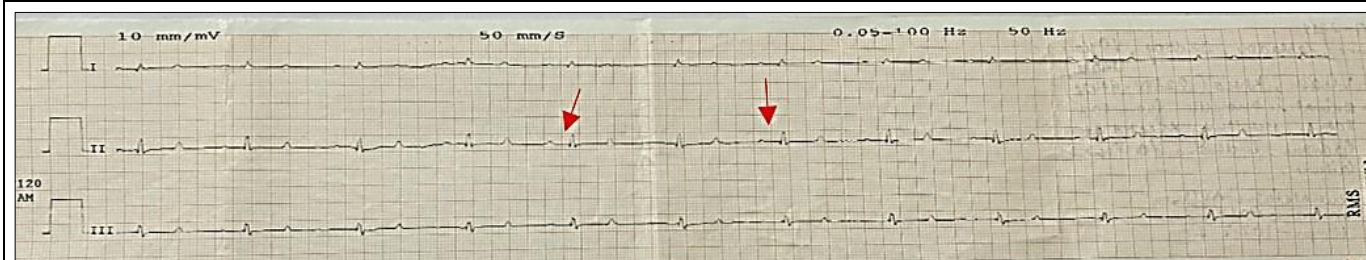


Fig 7: Pre-treatment ECG showing low QRS complexes ($<0.5\text{mV}$ amplitude) (50 mm/s paper speed; 10 mm/mV gain).

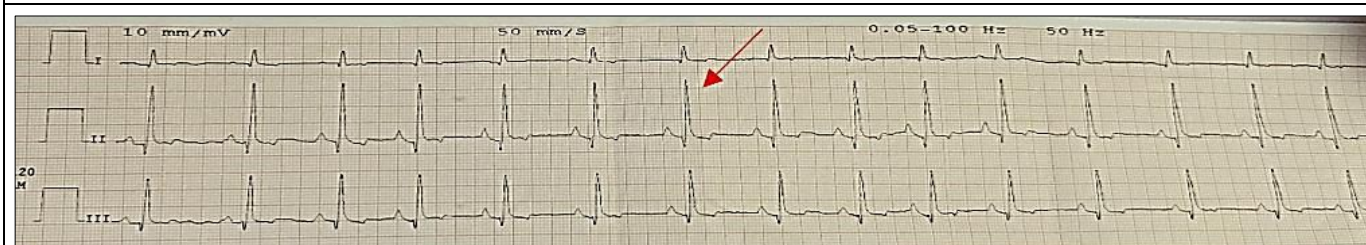


Fig 8: Improved amplitude of QRS amplitude after thoracocentesis (50 mm/s paper speed; 10 mm/mV gain).

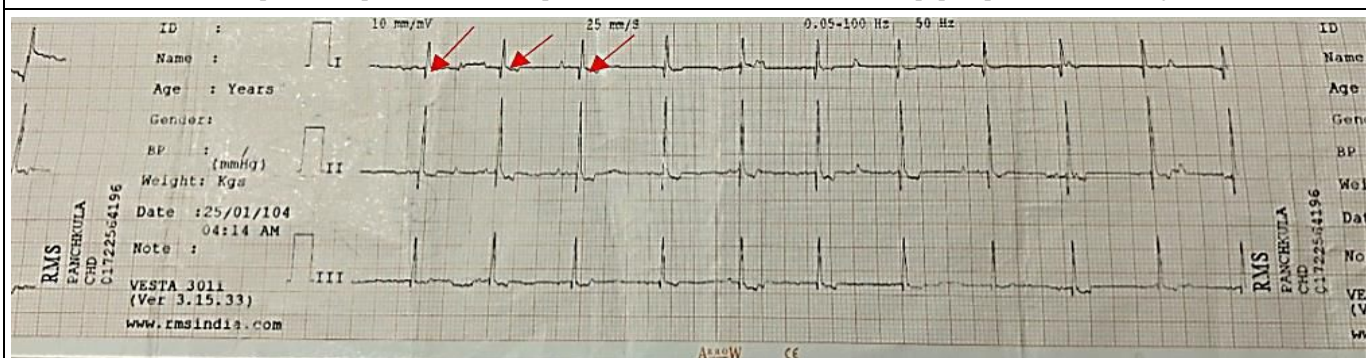


Fig 9: Pre-treatment ECG showing electrical alternans in a dog diagnosed with pericardial and pleural effusion (25 mm/s paper speed; 10 mm/mV gain).

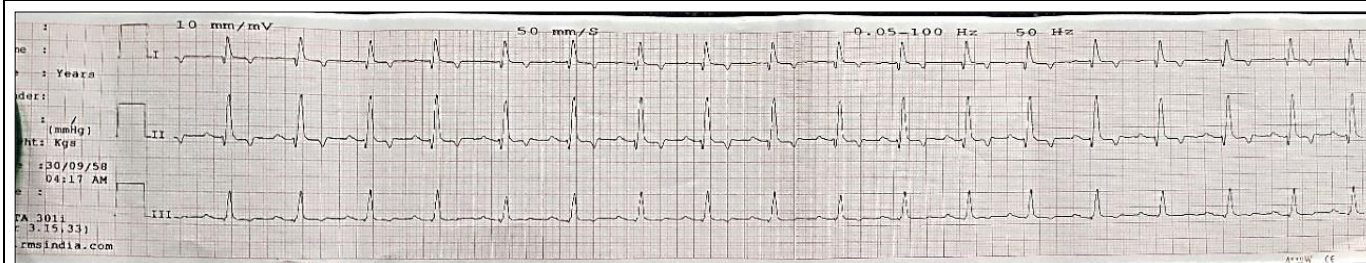


Fig 10: Normal ECG, showing no electrical alternans in a dog diagnosed with pericardial and pleural effusion after treatment with group II (50 mm/s paper speed ; 10 mm/mV gain).

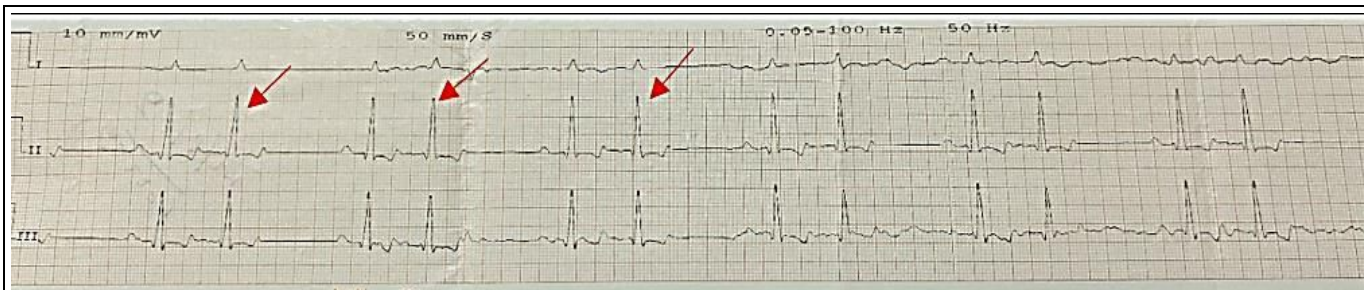


Fig 11: Pre-treatment ECG showing atrial premature complexes (APCs) in a dog diagnosed with severe DCM and severe mitral valve regurgitation (50 mm/s paper speed; 10 mm/mV gain).

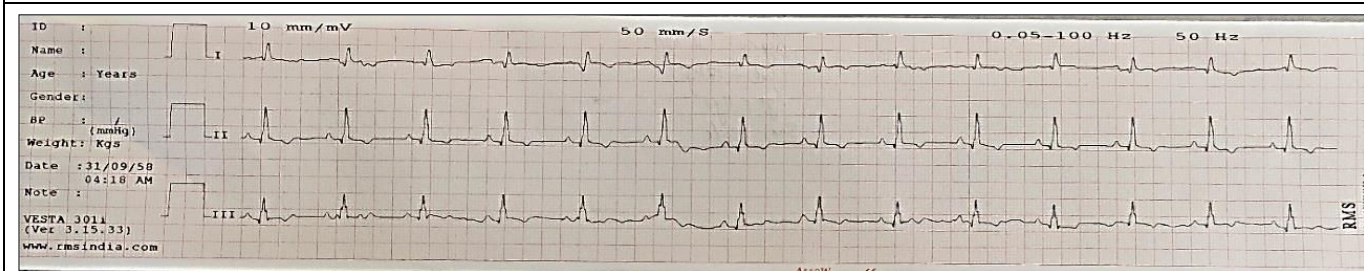


Fig 12: Post-treatment ECG showing no atrial premature complexes (APCs) after treatment with group I (50 mm/s paper speed; 10 mm/mV gain).

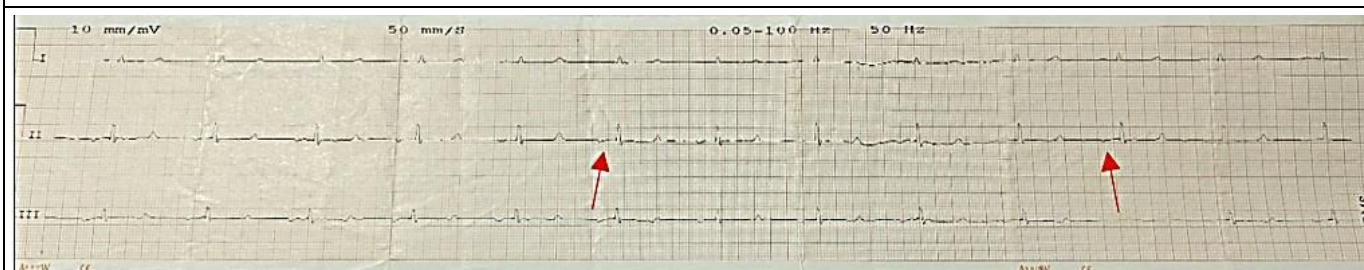


Fig 13: Negative p wave and normal QRS complexes (amplitude nearly 0.5 mV), suggestive of AV junctional premature complexes in HCM (50 mm/s paper speed; 10 mm/mV gain).

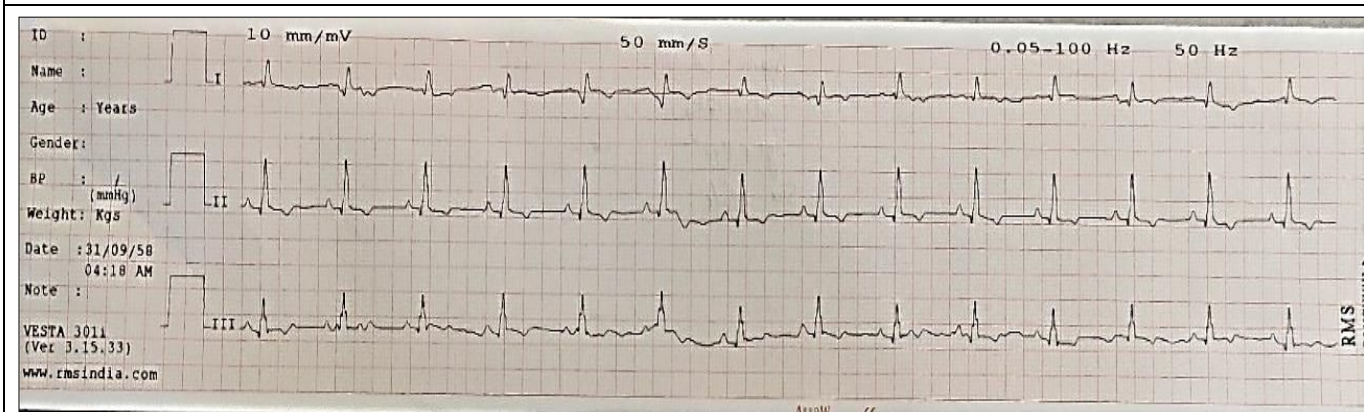


Fig 14: Post-treatment ECG showing recovery after treatment with group III (50 mm/s paper speed; 10 mm/mV gain).

Based on the comparison of cTn-I levels (pre- and post-treatment, with controls), survivability rates, pre and post treatments ECGs and the mean resolution of clinical signs, Group III (treated with carvedilol and pimobendane) demonstrated the most favourable outcomes. This group was especially effective in cases with severe tachycardia, severe hypertension, and atrial fibrillation. Group II (pimobendane) ranked second, while Group I (digoxin) showed the least favourable results.

Conclusion

The present study highlights the efficacy of different treatment protocols for managing congestive heart failure

(CHF) in geriatric dogs, focusing on their impact on clinical, haematological, and biochemical parameters. Group III, treated with a combination of carvedilol and pimobendane, demonstrated the most significant improvement in clinical outcomes, including normalization of haematological and biochemical parameters and a highly significant reduction in cardiac troponin-I (cTn-I) levels. This group also showed the highest survival rate and better resolution of clinical signs, underscoring the combined therapy's superiority in addressing severe cases of CHF, such as those with tachycardia, hypertension, and atrial fibrillation. The therapeutic benefits of carvedilol in mitigating adverse cardiac remodeling and stabilizing ventricular parameters,

alongside pimobendane's inotropic and vasodilatory effects, were evident. Group II, treated with pimobendane alone, exhibited moderate efficacy, while Group I, receiving digoxin, showed the least favourable outcomes. These findings emphasize the importance of tailored treatment approaches based on disease severity, with combined carvedilol and pimobendane therapy emerging as the most effective protocol for improving clinical outcomes and survival in geriatric dogs with CHF.

While this study offers valuable insights in management of CHF in dogs, it had certain limitations that warrant consideration. These include a small sample size, a short study duration, and variability in owner compliance and comorbidities. Advanced imaging techniques, such as echocardiography and radiography, were utilized for diagnosis but not for post-treatment evaluation. To build on these findings, future research should focus on larger cohorts, extended study periods, and comprehensive post-treatment assessments using advanced diagnostic tools.

Reference

1. Aiello SE, Moses DG, Allen PD, Constable A, Dart PR, Davies KE. Traumatic reticuloperitonitis. In: The Merck Veterinary Manual. 11th ed. Kenilworth, NJ: Merck & Co., Inc.; c2016. p. 230-3.
2. Ando T, Izawa T, Nishida H, Akiyoshi H. Clinical findings using echocardiography and plasma cardiac troponin I and pathological findings in dogs with hypertrophic cardiomyopathy: A retrospective study. *Open Vet J*. 2023;13(6):742-52.
3. Avila MS, Ayub-Ferreira SM, de Barros Wanderley MR, das Does Cruz F, Goncalves Brandao SM, Rigaud VO. Carvedilol for prevention of chemotherapy-related cardiotoxicity: The CECCY trial. *J Am Coll Cardiol*. 2018;71(20):2281-90.
4. Beaumier A, Rush JE, Yang VK, Freeman LM. Clinical findings and survival time in dogs with advanced heart failure. *J Vet Intern Med*. 2018;32:944-50.
5. Brložnik M, Pečjak A, Svete AN, Petrič AD. Selected hematological, biochemical, and echocardiographic variables as predictors of survival in canine patients with myxomatous mitral valve disease and congestive heart failure. *J Vet Cardiol*. 2023;46:18-29.
6. Deepti BR, Yathiraj S, Ramesh PT, Ranganath L, Narayanaswamy HD. Diagnosis and treatment of congestive heart failure in canines. *Intas Polivet*. 2016;17:129-31.
7. Dzau VJ, Colucci WS, Hollenberg NK, Williams GH. Relation of the renin-angiotensin-aldosterone system to clinical state in congestive heart failure. *Circ*. 1981;63(3):645-51.
8. Haggstrom J, Boswood A, O'Grady M. Effect of pimobendan or benazepril hydrochloride on survival times in dogs with congestive heart failure caused by naturally occurring myxomatous mitral valve disease: the QUEST study. *J Vet Intern Med*. 2008;22:1124-35.
9. Hoque M, Saxena AC, Gugjoo MB, Bodh D. Cardiac diseases in dogs. *Indian J Anim Health*. 2019;58(1):1-20.
10. Macdonald PS, Keogh AM, Aboyou CL, Lund M, Amor R, McCaffrey DJ. Tolerability and efficacy of carvedilol in patients with New York Heart Association Class IV heart failure. *J Am Coll Cardiol*. 1999;33(4):924-31.
11. Martin MW, Johnson MJ, Celona BJ. Canine dilated cardiomyopathy: A retrospective study of signalment, presentation, and clinical findings in 369 cases. *J Small Anim Pract*. 2009;50:23-29.
12. Mizuno M, Yamano S, Chimura S, Hirakawa A, Takusagawa Y, Sawada T. Efficacy of pimobendan on survival and reoccurrence of pulmonary edema in canine congestive heart failure. *J Vet Med Sci*. 2017;79(1):29-34.
13. Olsen LH, Häggström J, Petersen HD. Acquired valvular heart disease. In: Ettinger SE, Feldman FC, editors. *Textbook of Veterinary Internal Medicine*. 7th ed. Philadelphia: W.B. Saunders Co.; c2010. p. 1299-319.
14. Palazzuoli A, Quatrini I, Vecchiato L, *et al*. Left ventricular diastolic function improvement by carvedilol therapy in advanced heart failure. *J Cardiovasc Pharmacol*. 2005;45:563-8.
15. Reece WO. *Dukes' Physiology of Domestic Animals*. 12th ed. Ithaca: Cornell University Press; c2004. p. 999.
16. Reynolds C, Oyama MA. Biomarkers in the diagnosis of canine heart disease. *Vet Focus*. 2008;18(3):2-6.
17. Ristic J. Clinical assessment of the dog with suspected cardiac disease. In *Pract*. 2004;26:192-9.
18. Sharma P, Palahania A. Electrocardiography in canine cardiac diseases. *Trad Mod Vet Med*. 2023;1(14):17-22.
19. Soares EC, Pereira GG, Petrus LC, Neto ML, Yamaki FL, Larsson MHMA. Survival and echocardiographic evaluation of dogs with idiopathic dilated cardiomyopathy treated with carvedilol. *Braz J Vet Anim Sci*. 2010;62(3):555-63.
20. Tangmahakul N, Orton EC, Surachetpong SD. Investigation of red blood cell and platelet indices in adult dogs suffering from myxomatous mitral valve disease with and without pulmonary hypertension. *Front Vet Sci*. 2023;10:1234768.
21. Thakuria P, Pallabi DN, Kalita A, Phukan B, Changkija DRTC, Dutta BC. Serum biochemical changes in arrhythmia in dogs. *Indian J Chem Sci*. 2018;6(3):3116-18.
22. Tidholm A. Survival in dogs with dilated cardiomyopathy and congestive heart failure treated with digoxin, furosemide, and propranolol: A retrospective study of 62 dogs. *J Vet Cardiol*. 2006;8(1):41-7.
23. Urfer SR, Kaerberlein TL, Mailheau S, Bergman PJ, Creevy KE, Promislow DE. A randomized controlled trial to establish effects of short-term rapamycin treatment in 24 middle-aged companion dogs. *GeroScience*. 2017;39(2):117-27.
24. Wess G, Schulze A, Butz V, Simak J, Killich M, Keller LJM. Prevalence of dilated cardiomyopathy in Doberman Pinschers in various age groups. *J Vet Intern Med*. 2010;24:533-38.