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CASE REPORT

Acute hemodynamic and echocardiographic responses to pimobendan versus levosimendan in a dog with decompensated dilated cardiomyopathy

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Abstract

Dilated cardiomyopathy (DCM) is a major cause of systolic heart failure in dogs, characterized by ventricular dilation and reduced contractility. During acute decompensation, rapid inodilator therapy is essential to stabilize hemodynamics. This case describes serial changes in non-invasive blood pressure and left ventricular systolic function observed during sequential administration of two inodilator agents via different routes on consecutive days. An Anatolian shepherd dog was presented with acute respiratory distress. Echocardiography revealed severe left ventricular dilation and systolic dysfunction, and DCM was diagnosed using established criteria. Because oral therapy was initially not tolerated, intravenous pimobendan was administered, producing a rapid but transient improvement in systolic indices and global longitudinal strain (GLS). After transition to oral pimobendan the following day, systolic function improved mildly, while GLS remained markedly reduced. Levosimendan was subsequently administered as an intravenous bolus, resulting in only minimal improvement in systolic parameters and GLS. A continuous infusion was then initiated; however, systolic measurements remained largely unchanged during infusion. Serial echocardiographic assessment, particularly GLS, documented temporal changes in myocardial function during sequential inodilator therapy. These findings represent descriptive clinical observations from the management of a single patient and should not be interpreted as evidence of comparative efficacy or superiority of one inodilator over another. Because atrial fibrillation and temporal variation in heart rate may have influenced echocardiographic measurements throughout the monitoring period, the findings should be interpreted within the methodological limitations of this sequential single-case report.

Keywords Dilated cardiomyopathy · systolic dysfunction · pimobendan · levosimendan · global longitudinal strain · dogs

Background

Dilated cardiomyopathy (DCM) is a common myocardial disease in dogs and is characterized by progressive dilation of the ventricular chambers, thinning of myocardial walls, and declining systolic contractility (Simpson et al. 2021). These alterations result in reduced cardiac output and the activation of compensatory neurohormonal pathways, often culminating in congestive heart failure (CHF), arrhythmias, and a guarded prognosis (Nelson and Couto 2019; Simpson et al. 2021). Although the condition classically affects large and giant breeds, DCM may occur in dogs of various genetic backgrounds and can arise from numerous etiologies, including hereditary predisposition, nutritional imbalances, myocarditis, or idiopathic causes (Bonagura and Visser 2022; Nelson and Couto 2019; Simpson et al. 2021).

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Diagnosis has long been guided by the scoring system established by the European Society of Veterinary Cardiology (ESVC), which incorporates major and minor echocardiographic and electrocardiographic criteria to define pathological chamber enlargement and systolic dysfunction (Dukes-McEwan et al. 2003). Recent refinements in the morphological definition of DCM emphasize breed-adjusted reference intervals, changes in left ventricular (LV) geometry, and more objective systolic indices. Within this framework, LV global longitudinal strain (GLS) derived from two-dimensional speckle-tracking echocardiography (2D STE) has gained increasing prominence. GLS is more sensitive than conventional indices such as fractional shortening (FS) and ejection fraction (EF), particularly in differentiating early or subtle systolic impairment that may precede overt chamber dilation (Hamabe et al. 2021; Wess 2022). Consequently, GLS has become a valuable adjunct in both suspected early-stage DCM and in monitoring treatment response (Varlik et al. 2026).

Management of DCM centers on improving systolic function, reducing preload and afterload, controlling arrhythmias, and alleviating signs of CHF (Nelson and Couto 2019; Simpson et al. 2021). Pimobendan and levosimendan are both calcium-sensitizing inodilators that enhance myocardial contractility while exerting vasodilatory effects through complementary pharmacological mechanisms. Pimobendan additionally inhibits phosphodiesterase III (PDE-III), whereas levosimendan produces vasodilation primarily through activation of ATP-sensitive potassium (K_{ATP}) channels (Papp et al. 2006; Boyle and Leech 2012; Bell et al. 2016). Pimobendan is widely used in both chronic and acute settings. The oral formulation provides peak hemodynamic effect 2–4 h after administration, with measurable plasma levels lasting 8–12 h (Boyle and Leech 2012; Simpson et al. 2021). Recently available intravenous (IV) pimobendan shows much faster onset of inotropy, with rapid increases in LV contractility and reductions in filling pressures and systemic vascular resistance (Pichayapaiboon et al. 2021). This rapid onset makes IV pimobendan advantageous in emergency settings where oral absorption may be delayed. Levosimendan has been extensively used in human cardiology for acute systolic failure (Pashkovetsky et al. 2019). Experimental and limited clinical canine studies demonstrate beneficial effects on contractility, diastolic function, and myocardial efficiency without increased oxygen consumption (Pagel et al. 1997; Papp et al. 2006). However, its clinical utility in naturally occurring canine DCM remains insufficiently characterized.

Given these considerations, this case aims to characterize the short-term echocardiographic and hemodynamic changes observed during sequential administration of IV pimobendan followed by levosimendan as part of the clinical management of a dog with acute decompensated DCM. Owing to the sequential single-case design, the report is intended to describe temporal clinical observations rather than compare the efficacy of the two inodilators.

Case presentation

A 5-year-old, 50 kg male Anatolian Shepherd Dog presented with acute respiratory distress and hindlimb weakness. Physical examination revealed cyanosis, tachycardia (202 bpm), and tachypnea (104 rpm). Bloodwork showed nonspecific abnormalities (Supplementary Table A). Thoracic radiographs revealed generalized cardiomegaly with pleural effusion. ECG identified atrial fibrillation (AF), characterized by an irregularly irregular rhythm, absence of discernible P waves, and a rapid ventricular response.

Echocardiography (2D; M-mode; color, and CW/PW Doppler; Tissue Doppler Imaging; Tissue Tracking; and 2D STE) was performed using standard views (Vivid S60N Ultra Edition, GE Healthcare, Norway), as performed in our previous study (Varlik et al. 2026). At each time point, heart rate was documented, and echocardiographic measurements were averaged over three consecutive cardiac cycles to minimize beat-to-beat variability associated with AF. Serial heart rate measurements obtained throughout the study are presented in Supplementary Table B. In the present case, all echocardiographic apical views (2-chamber, 4-chamber, and apical long-axis) were acquired with frame rates exceeding 90 frames/s, consistent with veterinary 2D STE practice for reliable temporal resolution (Mogensen et al. 2025). Tracking quality was assessed visually, and regions of interest were manually adjusted until optimal border delineation was achieved; segments with poor tracking were excluded from analysis. Segment acceptance/rejection criteria were applied systematically, and segments failing quality control were omitted from GLS calculation. In instances where individual sequences exhibited segment loss, suboptimal frame rate, or excessive heart rate variability causing automatic AFI software failure or errors, measurements were obtained from alternative sequences within the same acquisition set to identify the optimal segment and cardiac cycle for analysis. All measurements were initially performed by a single operator (TV), and subsequently reviewed offline in the EchoPac module (GE Healthcare, Version 204, Norway) by a second cardiology-experienced investigator (ZY) to confirm results and minimize operator-dependent variability. This approach ensured that measurement reproducibility was optimized despite the single-case study design. A recent prospective study in dogs systematically evaluated 2D STE acquisition and analysis, reporting intra- and inter-observer coefficients of variation below 9%, supporting

the reliability of GLS measurements when acquisition and tracking quality are carefully controlled (Mogensen et al. 2025). Findings included marked LV dilation, low FS (18%), low EF (37%), increased EPSS, increased sphericity index, atrial enlargement, and AF. The ESVC score totaled 11, confirming DCM (Bonagura and Visser 2022) (Fig. 1). Because the dog exhibited severe systolic dysfunction with acute decompensation, immediate inodilator support was required. Initial supportive management included oxygen supplementation and furosemide (2 mg/kg, IV) to alleviate congestive signs. At presentation, the dog was unable to tolerate oral medication due to respiratory distress and clinical instability; therefore, an injectable formulation of pimobendan was selected as the initial emergency inodilator. Pimobendan (Vetmedin® 0.75 mg/ml Flacon, Boehringer Ingelheim, Germany) was administered intravenously at the dose of 0.15 mg/kg after baseline echocardiography to provide rapid inotropic support. A standardized echocardiographic measurement protocol was applied throughout all treatment phases, with assessments performed at predefined time points (15, 30, 60, and 120 min, and 24 h). Serial echocardiography was intentionally performed to capture rapid, short-term myocardial responses to inodilator therapy and to guide real-time clinical decision making. All examinations were time-limited, performed by the same experienced operator, and conducted only after stabilization of respiratory status to minimize patient stress. Following IV pimobendan administration (0.15 mg/kg), GLS improved from −10.6% at admission to −18% at 30 min (Figs. 1 and 2). FS and EF increased to 36% and 50%, respectively, before partially declining toward baseline values at 24-hour reassessment. Left ventricular outflow tract velocity time integral (LVOT VTI) increased from 10.3 cm at admission to 11.8 cm at 60 min and remained above baseline during the early post-treatment period (Fig. 2). Heart rate decreased from 200 bpm at baseline to a nadir of 130 bpm at 120 min before increasing to 160 bpm at the 24-hour reassessment (Supplementary Table B).

After completion of the initial intravenous stabilization and the 24-hour reassessment, the dog was able to tolerate

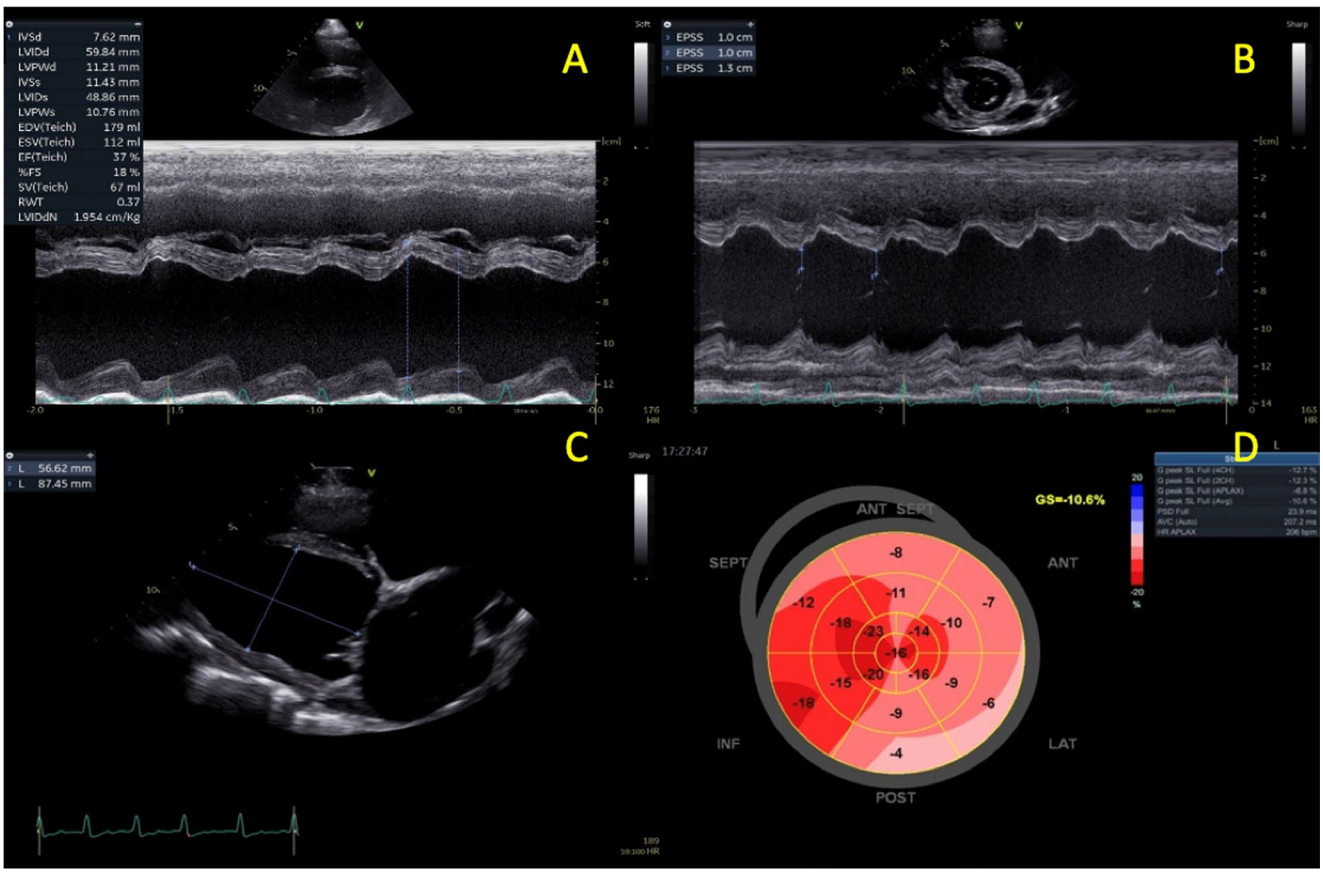

Fig. 1 Echocardiographic measurements were obtained at admission (pre-treatment). (**A**) Two-dimensional M-mode imaging from the RPLAx view showed left ventricular (LV) dilation with markedly reduced ejection fraction (EF: 37%) and fractional shortening (FS: 18%), consistent with cardiomegaly and systolic dysfunction indicative of dilated cardiomyopathy (DCM). (**B**) E-point septal separation (EPSS) measurements (≥1 cm) and (**C**) the sphericity index (1.54) further supported the diagnosis of DCM based on geometric remodeling. (**D**) Bull's eye strain map derived from apical two-, three-, and four-chamber views demonstrates severely reduced global longitudinal strain (GLS: -10.6%), confirming severe LV global systolic impairment

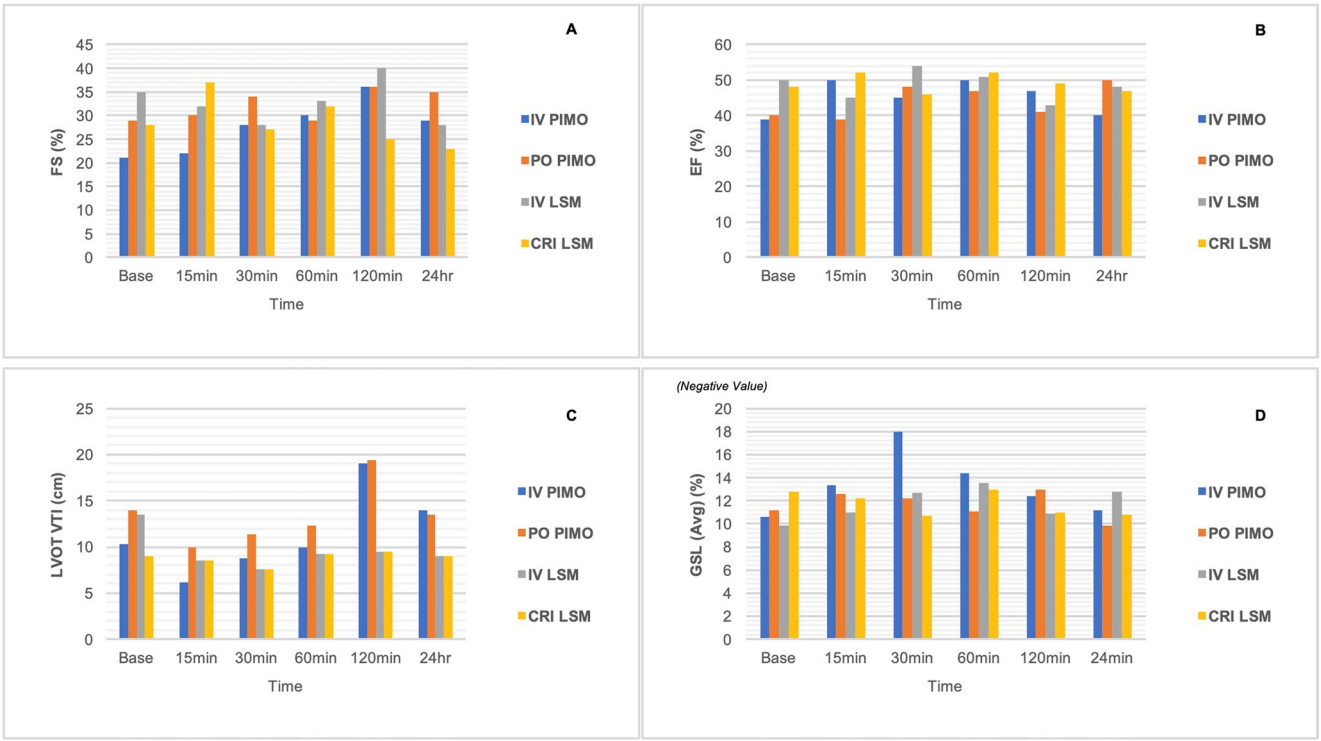

Fig. 2 Serial echocardiographic changes in systolic function and stroke volume indices following intravenous (IV) and oral pimobendan (PIMO), and IV bolus and continuous rate infusion (CRI) of levosimendan (LSM) in a dog with acute decompensated dilated cardiomyopathy (DCM). Measurements were obtained at baseline and at specified time points after each treatment phase. (**A**) FS%, (**B**) EF, (**C**) LVOT VTI, and (**D**) GLS were measured at baseline and at 15, 30, 60, and 120 min, and 24 h after each treatment phase. IV PIMO was administered on Day 1, followed by oral PIMO on Day 2. LSM was administered as an IV bolus on Day 3 and as a bolus plus CRI on Day 4. The figure illustrates the temporal evolution of echocardiographic variables during the sequential clinical management of this single patient

oral medication and was transitioned to oral pimobendan at a single dose of 0.5 mg/kg (Vetmedin® 10 mg, Boehringer Ingelheim, Germany). Although the recommended maintenance dosage of pimobendan is generally 0.25–0.30 mg/kg administered twice daily, the objective in this case was to provide continued inodilator support during the transition from emergency intravenous treatment to oral therapy rather than to establish long-term maintenance treatment. A single oral dose of 0.5 mg/kg was therefore selected to achieve prompt systemic drug exposure during this transitional phase. This approach was supported by pharmacokinetic studies demonstrating rapid absorption of pimobendan and formation of its active metabolite, o-desmethyl-pimobendan (ODMP), following oral administration in dogs (Her et al. 2020). Serial echocardiographic assessments were repeated according to the predefined protocol (Fig. 2). At the 24-hour reassessment, the initial echocardiographic improvements were only partially maintained, with recurrent reduction in systolic indices and GLS. During oral pimobendan administration, LVOT VTI values remained between 14.0 and 19.4 cm (Fig. 2). Heart rate remained persistently elevated during this treatment phase (180–208 bpm).

Because the initial improvement in myocardial performance was not maintained and no additional IV pimobendan was available, continued inodilator support was considered necessary. Accordingly, sequential treatment with levosimendan was initiated as the next therapeutic step. On Day 3, a levosimendan was administered as an intravenous bolus (24 µg/kg; Simdax 2.5 mg/ml, 5 ml flacon, Daiichi Sankyo Ltd, Istanbul, Türkiye), representing the first phase of the levosimendan treatment protocol. Because standardized evidence-based dosing recommendations for naturally occurring canine DCM are currently unavailable, the selected dosing regimen was based on previously published experimental studies in conscious dogs evaluating the acute hemodynamic effects of levosimendan (McGough et al. 1997; Kersten et al. 2000). Serial echocardiographic assessments were performed at predefined time points following bolus administration (Fig. 2). The 24-hour reassessment demonstrated persistent systolic dysfunction, with echocardiographic variables remaining within the range observed during this treatment phase.

Given the published evidence suggesting that continuous infusion may provide more sustained hemodynamic support, a second levosimendan administration was performed on Day

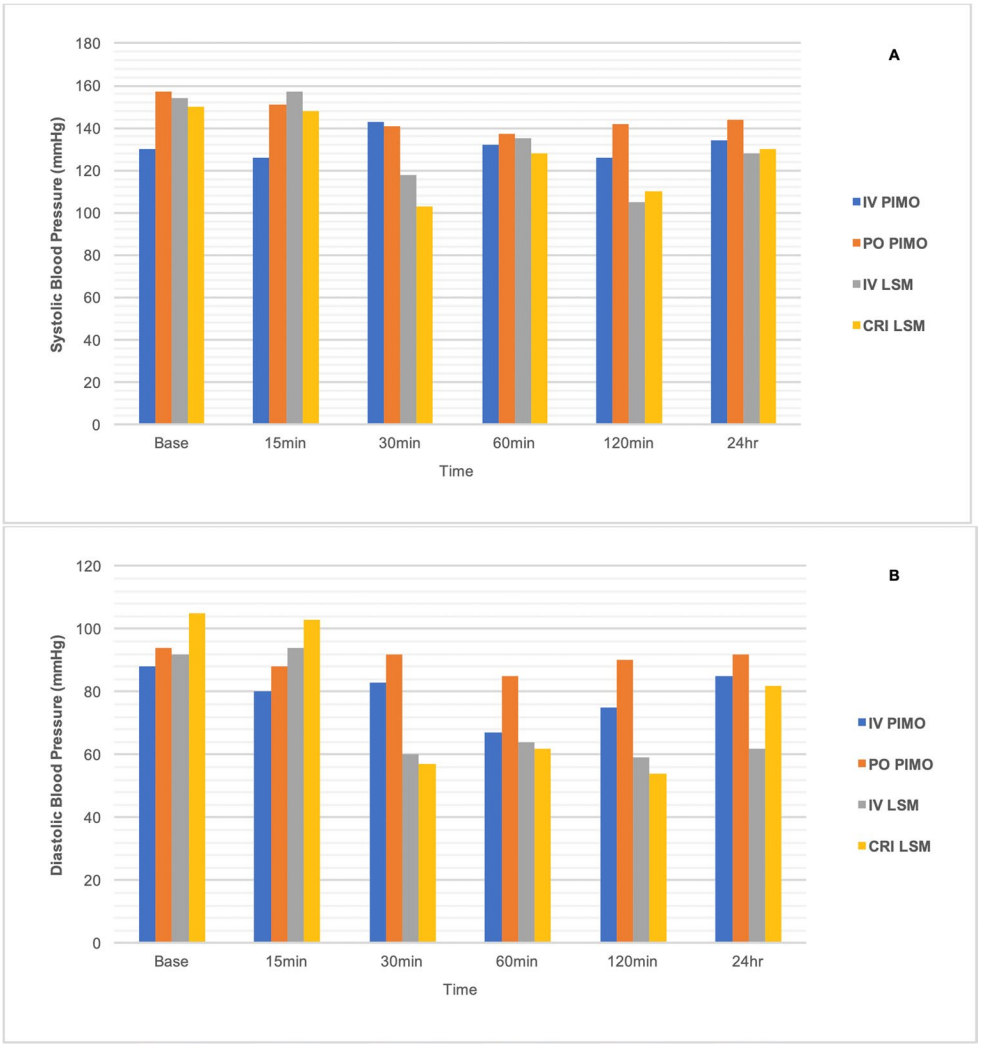

Fig. 3 Serial non-invasive systolic (**A**) and diastolic blood pressure measurements (**B**) following intravenous (IV) and oral pimobendan (PIMO), and IV bolus and continuous rate infusion (CRI) of levosimendan (LSM) in a dog with acute decompensated dilated cardiomyopathy (DCM). Treatment interventions and the timing of subsequent assessments were defined according to the protocol outlined in Fig. 2. The figure illustrates the temporal changes in non-invasive blood pressure during the sequential treatment phases in the clinical management of this single patient. These observations are presented descriptively and should not be interpreted as evidence of comparative efficacy or superiority of one inodilator over another

4 using an IV bolus immediately followed by CRI (0.4 µg/kg/min). Serial echocardiographic assessments were performed according to the predefined protocol after initiation of the 30-minute CRI (Fig. 2). Following bolus administration, FS reached 40%, and GLS was −13.5% at 60 min. LVOT VTI was measured at 13.5 cm before treatment and ranged between 8.5 and 9.5 cm during the subsequent bolus and early CRI period. No further improvement in systolic indices was observed during CRI (Fig. 2). Heart rate remained persistently elevated throughout the levosimendan treatment phase, ranging from 169 to 200 bpm (Supplementary Table B).

Non-invasive blood pressure (NIBP), oxygen saturation, heart rate (Supplementary Table B), and respiratory rate were recorded at each echocardiographic time point throughout all phases of treatment. NIBP remained stable during IV and oral pimobendan administration. During levosimendan bolus and early infusion, mild transient decreases in systolic and mean arterial pressures were recorded (Fig. 3).

Following completion of the levosimendan treatment protocol, the patient remained hospitalized for an additional two days. Rate-control therapy was intentionally deferred during the initial stabilization period because the primary therapeutic objective was rapid hemodynamic stabilization and improvement of forward cardiac output in the setting of acute decompensated systolic heart failure. Accordingly, the sequential inodilator protocol was concluded before ventricular rate-control therapy was initiated. Once the patient's clinical condition had stabilized, diltiazem and digoxin were introduced for ventricular rate control. Oral furosemide, pimobendan, diltiazem, and digoxin were administered during the hospitalization period, with adjustments based on the patient's clinical status. The patient showed progressive clinical improvement, with resolution of respiratory distress, normalization of activity level, and stabilization of vital signs. The dog was discharged in a stable and ambulatory condition. Follow-up communication with the owners one-week post-discharge indicated that the patient was in good general condition. However, the dog was not presented for re-examination, and therefore, long-term follow-up data are not available.

Discussion and conclusion

This case provides a detailed temporal description of echocardiographic and hemodynamic changes observed during sequential inodilator therapy in a dog with acute decompensated DCM. Because the therapeutic interventions were introduced sequentially in a single patient, these observations should not be interpreted as a direct comparison of treatment efficacy. Conventional echocardiographic indices such as FS and EF demonstrated relatively limited temporal variation during the sequential treatment phases, indicating restricted sensitivity for identifying rapid changes in myocardial performance in advanced disease. Similar observations have been reported previously, where automated or M-mode derived EF failed to correlate with myocardial contractile alterations detected by strain imaging (Suzuki et al. 2012).

In contrast, GLS exhibited substantial responsiveness to inodilator administration and more closely reflected treatment-associated changes in myocardial performance. However, the presence of AF represents an important confounding factor when interpreting systolic indices such as FS, EF, and GLS. Elevated heart rate may reduce diastolic filling time and alter loading conditions, potentially leading to underestimation of true myocardial contractility. In addition, AF introduces substantial beat-to-beat variability and irregular RR intervals, which may influence ventricular filling, stroke volume, and strain-derived parameters. Although averaging a higher number of cardiac cycles is generally recommended under such conditions, several human studies and consensus documents indicate that averaging at least three consecutive cardiac cycles can provide a reliable estimate. For example, Schach et al. (2021) acquired 3–5 consecutive cycles in AF when measuring LV volumes and strain, while the EACVI/ASE consensus document recommends that "measurements obtained from at least three consecutive cardiac cycles should be averaged" in AF (Badano et al.2018). In the present case, three consecutive cycles were analyzed in line with these approaches; however, this should still be considered a limitation, as averaging a greater number of cycles or applying an index-beat selection strategy based on similar RR intervals could have further improved measurement reliability. Furthermore, no index-beat selection was performed, and therefore, residual variability related to cycle length may have affected GLS measurements.

Heart rate remained persistently elevated throughout the monitoring period despite clinical management, with only partial reduction (lowest value: 154 bpm) (Supplementary Table B). Because ventricular rate-control therapy was intentionally postponed until completion of the initial inodilator protocol, persistent rapid ventricular response may have contributed to the temporal changes observed in FS, EF, GLS, and LVOT VTI in addition to the effects of the administered inodilators. The relative contribution of heart rate and the administered inodilators cannot be determined in this sequential single-case design and therefore represents an important limitation when interpreting the echocardiographic findings.

During the initial stabilization period, GLS improved after intravenous pimobendan administration, consistent with the reported rapid onset of action intravenous pimobendan (Pichayapaiboon et al. 2021). Subsequent echocardiographic evaluations performed during the later sequential treatment phases documented distinct temporal patterns of change. Accordingly, these findings should be interpreted as descriptive clinical observations rather than evidence of comparative efficacy between the administered inodilators. The partial decline observed at the 24-hour assessment is consistent with the short duration of the acute pharmacodynamic effects of intravenous pimobendan (Bell et al. 2016), supporting the interpretation that the initial improvement following IV administration may not be sustained without continued or repeated inotropic support.

Following completion of the emergency intravenous stabilization phase, a single oral dose of 0.5 mg/kg pimobendan was administered to provide continued inodilator support during the transition to oral therapy rather than to establish conventional maintenance treatment. Although this dosing strategy differs from the recommended maintenance regimen of 0.25–0.30 mg/kg administered twice daily (Boyle and Leech 2012; Bell et al. 2016), a single oral dose of 0.5 mg/kg has previously been used in pharmacokinetic studies in dogs to characterize systemic exposure to pimobendan and its active metabolite, o-desmethyl-pimobendan (Her et al. 2020). To maintain methodological consistency across the sequential treatment phases, each intervention was evaluated after a single administration using identical predefined monitoring intervals. Administering oral pimobendan according to the conventional twice-daily regimen would have introduced overlap between successive doses and complicated interpretation of the temporal echocardiographic observations. Accordingly, a single oral dose was selected to preserve a comparable observation window across the sequential treatment phases while maintaining continuous clinical inodilator support. Pimobendan has consistently been shown to improve myocardial contractility and hemodynamic function in dogs through calcium sensitization and PDE-III inhibition (Suzuki et al. 2011; Boyle and Leech 2012; Bell et al. 2016). The temporal echocardiographic changes observed during the oral pimobendan phase may therefore reflect a combination of the transitional single-dose administration, its pharmacokinetic profile (Boyle and Leech 2012), the reduced myocardial reserve

characteristic of advanced DCM (Varlik et al. 2026), and the evolving clinical condition of the patient. Consequently, the relative contributions of the dosing strategy, drug pharmacokinetics, disease severity, and changing loading conditions cannot be distinguished within the constraints of this sequential single-case report.

Doppler-derived indices provided additional insight into forward flow dynamics in this case. The increase in LVOT VTI following intravenous pimobendan suggests an increase in forward flow under the prevailing hemodynamic conditions, whereas the relatively higher LVOT VTI values during oral administration compared to baseline indicate continued hemodynamic support despite only modest changes in conventional systolic indices. During the subsequent levosimendan treatment phases, LVOT VTI values differed from those observed during the preceding treatment phases. These temporal changes may have been influenced by multiple factors, including vasodilatory effects, loading conditions, atrial fibrillation, and the evolving clinical status of the patient. However, LVOT VTI was not combined with LVOT diameter in this case and therefore cannot be used as a direct surrogate for stroke volume. In addition, the presence of atrial fibrillation with tachycardia likely introduced beat-to-beat variability, and changes in VTI may partially reflect irregular cycle length rather than true alterations in stroke volume. Accordingly, LVOT VTI should be interpreted as a descriptive parameter reflecting relative changes in forward flow rather than a quantitative measure of stroke volume.

The introduction of levosimendan was pharmacologically motivated to provide continued inodilator support after the initial pimobendan treatment phase. Although both pimobendan and levosimendan share calcium sensitization as their principal positive inotropic mechanism, their ancillary pharmacological actions differ. Pimobendan additionally inhibits PDE-III, whereas levosimendan promotes vasodilation predominantly through activation of K_{ATP} channels (Papp et al. 2006; Boyle and Leech 2012; Bell et al. 2016). These complementary ancillary mechanisms provided the pharmacological rationale for the sequential therapeutic approach adopted in this case rather than representing fundamentally different inotropic mechanisms. Although experimental studies have demonstrated potent inodilator effects of levosimendan (Harkin et al. 1995), myocardial responsiveness may be reduced in advanced DCM, potentially limiting its clinical impact.

Echocardiographic indices such as EF, FS, and GLS are known to be load-dependent and may increase following reductions in preload, particularly in response to diuretic therapy (Suzuki et al. 2012; Bonagura and Visser 2022). In this case, furosemide was administered at presentation and would be expected to reduce ventricular filling pressures and wall stress, potentially leading to improvements in systolic and strain-derived parameters independent of intrinsic myocardial contractility (Nelson and Couto 2019). However, the temporal changes observed in GLS and conventional systolic indices are unlikely to be explained by loading conditions alone. Because preload, afterload, heart rate, concurrent medical therapy, and overall clinical status changed throughout hospitalization, the relative contributions of these factors and the administered inodilators cannot be determined in this sequential single-case design. The observed temporal changes are broadly compatible with the known pharmacodynamic properties of both inodilators. Although both drugs exert positive inotropic effects through calcium sensitization, ancillary mechanism−PDE III inhibition by pimobendan and K_{ATP}-sensitive channel activation by levosimendan−may contribute to differences in their hemodynamic profiles (Papp et al. 2006; Antila et al. 2007; Boyle and Leech 2012; Bell et al. 2016). Accordingly, the absence of standardized control of preload, afterload, and systemic vascular resistance represents an important methodological limitation, and the findings should be interpreted with appropriate caution (Bonagura and Visser 2022). Future studies under controlled loading conditions are warranted to better delineate the relative contributions of pharmacologic and hemodynamic factors.

The dosing regimen used in this case was selected according to previously published experimental canine studies; however, evidence-based dosing recommendations for naturally occurring canine DCM remain limited. Accordingly, the levosimendan protocol was based primarily on experimental studies in conscious dogs evaluating the acute hemodynamic effects of levosimendan (McGough et al. 1997; Kersten et al. 2000), as standardized veterinary clinical dosing guidelines are currently unavailable. Although levosimendan is widely used in human patients with acute heart failure and is included as a therapeutic option in current ESC heart failure guidelines (McDonagh et al. 2023), the dosing regimen employed in the present case was not directly extrapolated from human cardiology but was selected on the basis of the available experimental canine evidence. Therefore, the chosen protocol should be regarded as an empirically supported dosing strategy rather than a validated clinical regimen for naturally occurring canine DCM.

In human medicine, levosimendan is typically administered as a prolonged (typically 24-hour) continuous intravenous infusion, providing sustained exposure to the parent compound and its long-acting metabolite OR-1896 (McDonagh et al. 2023). Whether a similar extended-duration CRI could enhance clinical response in dogs remains unknown. In the present case, a short-duration infusion protocol was employed due to clinical constraints and the

need for rapid, repeated echocardiographic assessments in an acutely unstable patient. This abbreviated protocol may have limited the time available for the development of sustained pharmacologic effects and for the accumulation of active metabolites, which likely contributed to the modest and transient hemodynamic responses observed. These findings suggest that shorter CRI periods may be insufficient in canine DCM, and further prospective studies are warranted to establish evidence-based dosing recommendations and to determine whether extended-duration (e.g., 24-hour) CRI protocols could produce more pronounced or sustained improvements in myocardial performance (Harkin et al. 1995; Pashkovetsky et al. 2019).

Blood pressure trends observed during the sequential treatment phases were broadly compatible with the reported pharmacodynamic profiles of the administered inodilators. Pimobendan maintained relatively stable systolic and mean arterial pressures, whereas levosimendan was associated with mild transient reductions in arterial pressure. Although these findings are compatible with the known vasodilatory effects of levosimendan mediated through K_{ATP}-sensitive channel activation (Antila et al. 2007), alternative explanations, including changes in forward flow, evolving loading conditions, progression of myocardial dysfunction, and the overall clinical status of the patient, cannot be excluded. Because cardiac output, systemic vascular resistance, and other invasive hemodynamic variables were not measured, the mechanisms underlying the observed arterial pressure changes cannot be determined with certainty (Antila et al. 2007).

Taken together, the present case should be regarded as hypothesis-generating rather than hypothesis-confirming, providing preliminary clinical observations to guide future prospective studies.

In conclusion, serial echocardiographic assessment, particularly GLS, documented temporal changes in myocardial function during the sequential clinical management of this dog with acute decompensated DCM. Because the therapeutic interventions were administered sequentially in a single patient under evolving clinical conditions, these observations should not be interpreted as evidence of comparative efficacy or superiority of one inodilator over another. Rather, this case highlights the potential value of serial strain imaging for monitoring short-term changes in myocardial performance during intensive clinical management. Tachycardia-induced cardiomyopathy represents an important differential diagnosis in patients presenting with AF and severe systolic dysfunction. In this case, the possibility of tachycardia-mediated myocardial depression cannot be fully excluded. However, the presence of marked ventricular dilation, increased sphericity index, elevated EPSS, and fulfillment of established ESVC diagnostic criteria support a diagnosis of primary DCM rather than a purely rate-related cardiomyopathy. Nevertheless, a component of tachycardia-induced functional deterioration superimposed on underlying DCM remains possible. The temporal echocardiographic changes observed during sequential treatment phases likely reflect the combined influence of the pharmacodynamic properties of the administered inodilators, evolving hemodynamic conditions, atrial fibrillation, heart rate variation, advanced myocardial disease, and concurrent medical therapy, rather than isolated differences in drug efficacy. Accordingly, the use of three cardiac cycles without index-beat selection in the setting of AF and persistent tachycardia, together with the observed temporal variation in heart rate documented throughout hospitalization, should be considered a methodological limitation, and the GLS findings should be interpreted with appropriate caution. Prospective controlled studies are warranted to better characterize the shorth-term hemodynamic and echocardiographic effects of different inodilator strategies in dogs with DCM and to determine whether longer-duration levosimendan CRI protocols, similar to those used in human medicine, provide additional clinical benefit.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s11259-026-11511-x.

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Author contributions Z.Y., T.V., D.A.: Conceptualization, data collection, clinical evaluation, manuscript writing; S.S., R.T.: Supervision, manuscript review; E.P.: Clinical management, manuscript review.

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Data availability The raw data and datasets supporting the findings of this study, which are not explicitly included within the article, are available from the corresponding author upon reasonable request.

Declarations

Ethical approval Written informed consent was obtained from the owner for the clinical management and publication of this case.

Conflict of interest The authors declare that they have no conflicts of interest related to this publication.

Competing interests The authors declare no competing interests.

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