RESEARCH ARTICLE

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Impact of Digoxin Therapy on Cardiac Mechanical Dyssynchrony in Patients with Non-ischemic Heart Failure

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Abstract

Background and Aim: Mechanical dyssynchrony is a key determinant of impaired cardiac performance, adverse remodeling, and prognosis in heart failure (HF). Digoxin is widely used in HF because of its positive inotropic and neurohormonal effects; however, its impact on mechanical dyssynchrony remains poorly defined.

Materials and Methods: In this prospective, randomized, single-center study, 50 patients with newly diagnosed non-ischemic dilated cardiomyopathy, left ventricular ejection fraction (LVEF) <45%, and documented mechanical dyssynchrony by tissue Doppler imaging were randomized to guideline-directed medical therapy (GDMT) alone (control) or GDMT plus digoxin (0.125-0.25 mg/day). Echocardiographic parameters, including intraventricular and interventricular dyssynchrony, left ventricular volumes, LVEF, and QRS duration, were assessed at baseline and at three months.

Results: At three months, intraventricular dyssynchrony decreased significantly in both groups, with a greater reduction in the digoxin group ($P < 0.001$ within-group; $P = 0.044$ between-group). Interventricular dyssynchrony improved significantly only in the digoxin group ($P = 0.002$). Digoxin therapy was associated with increased LVEF ($P = 0.006$), reduced left ventricular volumes, and shorter QRS duration ($P < 0.001$). No significant changes were observed in the control group for these parameters.

Conclusion: Digoxin therapy appears to improve cardiac mechanical synchrony, systolic function, and electrical conduction in patients with non-ischemic HF. These findings suggest that the therapeutic effects of digoxin may extend beyond symptom relief and neurohormonal modulation to include favorable mechanical remodeling.

Keywords: Heart failure, imaging, left ventricular dysfunction

INTRODUCTION

Heart failure (HF) remains a major public health problem, associated with substantial morbidity, mortality, and healthcare burden.[1] Mechanical dyssynchrony—defined as a temporal mismatch in regional myocardial contraction—plays a critical role in the pathophysiology of HF and is associated with adverse ventricular remodeling, impaired hemodynamic efficiency, and worse clinical outcomes.[2,3] Intraventricular and interventricular dyssynchrony have been identified as independent predictors of poor prognosis in patients with HF, particularly those with reduced ejection fraction (HFrEF).[3]

Pharmacologic and device-based strategies targeting dyssynchrony have demonstrated beneficial effects on clinical outcomes. Beta-blockers have been shown to reduce mechanical dyssynchrony in selected patients with dilated cardiomyopathy,[4] while cardiac resynchronization therapy

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(CRT) has become a cornerstone therapy for patients with electrical dyssynchrony and advanced HF.[5,6] Despite these advances, many patients remain symptomatic or ineligible for device therapy, highlighting the need for adjunctive pharmacologic strategies.

Digoxin, a cardiac glycoside, has been used for decades in the management of HF, primarily for rate control in atrial fibrillation and for symptomatic improvement in HFrEF. Its mechanism of action involves inhibition of the Na^+/K^+-ATPase, resulting in increased intracellular calcium and enhanced myocardial contractility. Digoxin also exerts neurohormonal effects, including modulation of sympathetic activity and vagal tone, which may influence myocardial electrical and mechanical function.[7] Digoxin inhibits the Na^+/K^+-ATPase pump, thereby increasing intracellular calcium availability through the $Na^+/Ca2^+$ exchanger. Improved calcium cycling may reduce regional disparities in myocardial contraction, shorten electromechanical delay, and ultimately improve ventricular mechanical synchrony. These effects may also interact with autonomic modulation and with the reversal of electrical remodeling. However, the effect of digoxin on mechanical dyssynchrony has not been systematically evaluated. Given its electrophysiological and hemodynamic effects, we hypothesized that digoxin may reduce mechanical dyssynchrony in patients with non-ischemic HF. Although digoxin has gradually shifted from a first-line therapy to an adjunctive treatment in contemporary HF guidelines because of concerns regarding its narrow therapeutic window and arrhythmogenic potential, low-dose therapy remains recommended for selected patients for symptom control and reduction in hospitalizations for HF. Whether digoxin can improve ventricular mechanical synchrony beyond its established positive inotropic and neurohormonal effects has not been adequately investigated. Demonstrating such an effect could provide additional mechanistic insight into the role of digoxin in selected patients with non-ischemic HF. We hypothesized that low-dose digoxin therapy (0.125-0.25 mg/day), administered within the currently recommended therapeutic range, would improve mechanical synchrony by enhancing intracellular calcium handling and myocardial contractile coordination without increasing the risk of digoxin-related toxicity.

METHODS

This study was designed as a prospective, single-center, randomized controlled trial conducted between August 2016 and March 2017. The study protocol was reviewed and approved by the Local Ethics Committee of Erciyes University (decision no. 2016/381; date: 24 June 2016). All participants provided written informed consent in accordance with the principles of the Declaration of Helsinki.

A total of 312 patients presenting with newly diagnosed HF were screened. Patients were eligible if they were older than 18 years, had non-ischemic dilated cardiomyopathy, were in sinus rhythm, had left ventricular ejection fraction (LVEF) <45%, and had mechanical dyssynchrony documented by tissue Doppler imaging (TDI). All patients were treatment-naïve with respect to HF therapy. Patients were excluded if they had ischemic heart disease (defined as ≥50% stenosis in a major epicardial coronary artery on coronary angiography), significant valvular disease, prior cardiac surgery, prior CRT or permanent pacemaker implantation, or any contraindication to digoxin.

After applying the eligibility criteria, 50 patients were enrolled and randomized to two groups. The control group received guideline-directed medical therapy (GDMT), including angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, beta-blockers, and mineralocorticoid receptor antagonists, as appropriate. The digoxin group received digoxin in addition to GDMT; digoxin was initiated at 0.125 mg/day and titrated up to 0.25 mg/day according to clinical tolerance and renal function.

All patients underwent transthoracic echocardiography at baseline and after three months using standardized protocols. Echocardiographic assessments were performed by two experienced cardiologists blinded to treatment allocation. Left ventricular volumes and LVEF were calculated using the modified Simpson's biplane method. Mechanical dyssynchrony was assessed using pulsed-wave TDI. Intraventricular dyssynchrony was defined as a time difference >60 ms between peak systolic velocities of the basal septal and lateral or posterior segments.[8] Interventricular dyssynchrony was defined as a delay >56 ms between peak systolic velocities of the right ventricular free wall and left ventricular basal segments.[9]

Clinical and echocardiographic parameters, including LV end-diastolic and end-systolic volumes, LVEF, QRS duration, and dyssynchrony indices, were recorded at baseline and follow-up. In addition to within-group comparisons, analysis of covariance (ANCOVA) was performed to compare post-treatment echocardiographic parameters between groups after adjustment for baseline values. The treatment group was entered as the fixed factor, and the baseline measurements were entered as covariates.

Statistical Analysis

Statistical analyses were performed using standard software. Continuous variables are presented as mean ± standard deviation or median (interquartile range), as appropriate. Categorical variables are expressed as counts and percentages. Between-group comparisons were conducted using one-way analysis of variance or the Kruskal-Wallis test; post-hoc analyses were conducted using Tukey's test or the Bonferroni

correction, as applicable. Categorical variables were compared using the chi-square or Fisher's exact test. Correlations were assessed using Pearson or Spearman correlation coefficients. All statistical tests were two-tailed, and a *P*-value < 0.05 was considered significant. Statistical analyses were performed using SPSS software (version 25.0; IBM Corp., Armonk, NY, USA).

RESULTS

A total of 49 patients completed the study (25 in the digoxin group and 24 in the control group), while one patient withdrew from the study after randomization. Baseline demographic, clinical, and echocardiographic characteristics were comparable between the two groups, including age, sex distribution, baseline heart rate, blood pressure, QRS duration, left ventricular volumes, and baseline mechanical dyssynchrony indices. No statistically significant differences were observed between groups baseline LVEF, intraventricular dyssynchrony, interventricular dyssynchrony, or concomitant medical therapies. Detailed baseline demographic, clinical, echocardiographic, and pharmacological characteristics of the study population are presented in Table 1.

At the end of the three-month follow-up period, both groups exhibited reductions in intraventricular dyssynchrony; however, the magnitude of improvement was more pronounced in the digoxin group. Intraventricular dyssynchrony decreased from 76.56±18.35 ms to 63.68±16.94 ms in the digoxin group ($P < 0.001$), representing a clinically and statistically significant improvement. In the control group, intraventricular dyssynchrony decreased from 70.79±14.03 ms to 64.50±13.59 ms ($P = 0.013$). The between-group comparison demonstrated that the digoxin group experienced a significantly greater reduction ($P = 0.044$), suggesting a treatment-specific effect beyond that achieved with standard therapy alone.

Interventricular dyssynchrony followed a similar trend. Patients receiving digoxin demonstrated a significant reduction in interventricular dyssynchrony from 71.84±22.53 ms to 62.32±21.64 ms ($P = 0.002$), whereas the control group showed no significant change (69.50±21.14 ms to 66.63±18.36 ms; $P = 0.324$). This finding indicates that digoxin may exert specific effects on biventricular mechanical coordination, influencing interventricular timing and efficiency.

Left ventricular systolic function improved significantly in the digoxin group. LVEF increased from 36.81%±7.24% to 40.28%±7.85% ($P = 0.006$), whereas no significant change was observed in the control group (39.42%±6.89% to 39.98%±9.19%; $P = 0.577$). This improvement was accompanied by significant reductions in left ventricular end-diastolic volume (LVEDV) and left ventricular end-systolic volume (LVESV) in the digoxin group, consistent with favorable reverse remodeling. No significant volumetric changes were observed in the control group. After adjustment for baseline values using ANCOVA, the beneficial effects of digoxin remained significant. Adjusted post-treatment intraventricular dyssynchrony was significantly lower in the digoxin group than in the control group (adjusted mean difference -6.1 ms, F=4.42, $P = 0.041$). Likewise, interventricular dyssynchrony remained significantly reduced (F=6.31, $P = 0.015$). Adjustment for baseline LVEF confirmed that post-treatment LVEF was significantly higher in the digoxin

Table 1. Baseline demographic, clinical, echocardiographic characteristics and pharmacological therapies

Variable	Digoxin group (n=25)	Control group (n=24)	*P*-value
Age, years	61.2±10.4	60.5±11.2	0.812
Male sex, n (%)	17 (68.0)	16 (66.7)	0.919
Heart rate, bpm	78.4±9.6	77.8±10.1	0.829
Systolic BP, mmHg	118.6±12.4	120.1±11.8	0.665
Diastolic BP, mmHg	74.2±8.1	75.0±7.6	0.731
QRS duration, ms	127.1±15.2	125.8±16.1	0.773
LVEF, %	36.81±7.24	39.42±6.89	0.203
LVEDV, mL	168.4±32.6	165.7±30.8	0.761
LVESV, mL	106.2±25.4	101.8±24.1	0.535
Intraventricular dyssynchrony, ms	76.56±18.35	70.79±14.03	0.211
Interventricular dyssynchrony, ms	71.84±22.53	69.50±21.14	0.716
Beta-blocker therapy, n (%)	21 (84.0)	20 (83.3)	0.946
ACE inhibitor/ARB/ARNI, n (%)	23 (92.0)	22 (91.7)	0.966
Mineralocorticoid receptor antagonist, n (%)	18 (72.0)	17 (70.8)	0.924

Values are presented as mean ± standard deviation or n (%), as appropriate

BP: Blood pressure, LVEF: Left ventricular ejection fraction, LVEDV: Left ventricular end-diastolic volume, LVESV: Left ventricular end-systolic volume, ACE: Angiotensin-converting enzyme, ARB: Angiotensin receptor blocker, ARNI: Angiotensin receptor-neprilysin inhibitor

group (F=7.18, P = 0.010). Similar findings were observed for LVEDV (F=5.27, P = 0.026), LVESV (F=6.94, P = 0.011), and QRS duration (F=10.84, P = 0.002), indicating that the observed treatment effects were independent of baseline differences.

Furthermore, a significant reduction in QRS duration was observed in the digoxin group (from 127.08 ms to 117.96 ms; P < 0.001), while QRS duration remained unchanged in the control group. The temporal changes in mechanical dyssynchrony parameters and left ventricular systolic function during the three-month follow-up period are illustrated in Figure 1. This finding suggests a potential electrophysiological effect of digoxin that may contribute to improved mechanical synchrony. This finding suggests that the reduction in QRS duration may reflect secondary improvements in ventricular geometry, reverse remodeling, and autonomic modulation rather than a direct electrophysiological action.

DISCUSSION

This study provides important insights into the potential role of digoxin in modulating mechanical dyssynchrony in patients with non-ischemic HF. The findings demonstrate that digoxin therapy is associated with significant improvements in both intraventricular and interventricular dyssynchrony, as well as in systolic function, parameters of ventricular remodeling, and electrical conduction. Mechanical dyssynchrony is increasingly recognized as a key determinant of disease progression in HF. It contributes to impaired contractile efficiency, increased wall stress, and progressive ventricular remodeling, all of which are associated with adverse clinical outcomes.[2,3] The prognostic relevance of dyssynchrony has been highlighted in prior studies, which demonstrated that patients with significant mechanical dyssynchrony experience higher rates of hospitalization, ventricular arrhythmias, and mortality.[10,11]

The mechanisms underlying the observed improvements with digoxin are likely multifactorial. Digoxin exerts a positive inotropic effect through inhibition of Na^+/K^+-ATPase, leading to increased intracellular calcium and enhanced myocardial contractility.[7] Improved contractile performance may facilitate more synchronous contraction patterns, thereby reducing temporal dispersion of myocardial shortening. Additionally, digoxin has well-established neurohormonal effects, including attenuation of sympathetic overactivity and enhancement of parasympathetic tone. These autonomic effects may influence myocardial conduction velocity and electromechanical coupling, contributing to improved mechanical synchrony.[11-17]

The significant reduction in QRS duration observed in the digoxin group supports the hypothesis that digoxin may exert favorable effects on electrical conduction pathways. While digoxin is not traditionally considered a primary electrophysiological agent affecting ventricular conduction, its indirect effects on autonomic tone and intracellular calcium handling may promote more coordinated ventricular activation. Improved electrical synchrony may, in turn, translate into improved mechanical synchrony, creating a positive feedback loop that enhances overall cardiac performance.[12-14] The observed reduction in QRS duration following digoxin therapy should not necessarily be interpreted as a direct electrophysiological effect. Instead, improved ventricular synchrony may reflect

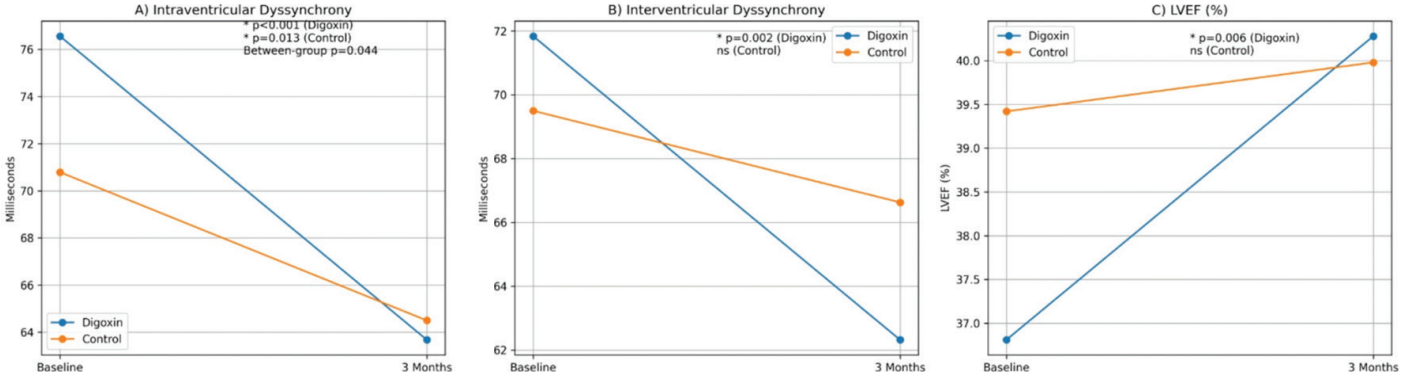

Figure 1. Changes in cardiac mechanical dyssynchrony and systolic function over three months

(A) Intraventricular dyssynchrony significantly decreased in both groups, with a greater reduction observed in the digoxin group (P < 0.001) compared with the control group (P = 0.013); the between-group difference was significant (P = 0.044)

(B) Interventricular dyssynchrony improved significantly only in the digoxin group (P = 0.002), while no significant change was observed in the control group

(C) Left ventricular ejection fraction (LVEF) increased significantly in the digoxin group (P = 0.006) with no significant change in the control group

**: Values are expressed as mean values, asterisks indicate statistical significance, ns: Not significant*

enhanced myocardial contractile coordination, mediated by improved intracellular calcium handling, enhanced autonomic modulation through increased vagal tone, and early reverse ventricular remodeling. These mechanisms may collectively contribute to a reduction in electromechanical delay, although further mechanistic studies are required.

The observed improvements in left ventricular volumes and LVEF suggest that digoxin may also contribute to reverse remodeling. Reverse remodeling is a critical therapeutic goal in HF management, as it is associated with improved functional capacity, reduced hospitalizations, and improved survival. The reduction in LV volumes observed in this study indicates a potential structural benefit associated with digoxin therapy, potentially complementing its functional effects.

Notably, the improvements observed in the digoxin group occurred in the context of GDMT, which included beta-blockers, renin-angiotensin system inhibitors, and mineralocorticoid receptor antagonists. This suggests that digoxin may provide incremental benefit when added to contemporary medical therapy.[11,17] In contrast, patients in the control group, who received guideline-directed therapy alone, demonstrated only modest improvements in mechanical parameters and no significant improvements in systolic function or remodeling indices. According to contemporary ESC and AHA/ACC HF guidelines, digoxin is recommended as an adjunctive therapy in selected patients who remain symptomatic despite optimal GDMT, rather than as a routine first-line treatment. Therefore, the present findings should be interpreted as providing mechanistic support for the use of digoxin in carefully selected patients rather than advocating its broader use.

The clinical implications of these findings are potentially important. While CRT remains the gold standard for patients with electrical dyssynchrony and wide QRS complexes, many patients with mechanical dyssynchrony do not meet criteria for device therapy. Pharmacologic strategies capable of improving mechanical synchrony could therefore have a meaningful impact on patient outcomes. The present findings raise the possibility that digoxin may serve as a useful adjunct in selected patients, particularly those with mechanical dyssynchrony who are not candidates for device-based therapies.

Study Limitations

This study has several limitations. This was a hypothesis-generating pilot trial, and the relatively small sample size limited statistical power and generalizability. First, the study was single-center, with a relatively small sample size and a short, three-month follow-up period, which limited statistical power and long-term generalizability. Second, mechanical dyssynchrony was assessed using TDI rather than speckle-tracking echocardiography; the latter may provide a more comprehensive assessment of myocardial mechanics. Although TDI is a well-established and widely available echocardiographic technique, it has technical limitations, including angle dependence and limited ability to assess global myocardial deformation compared with speckle-tracking echocardiography (STE). STE may provide a more comprehensive evaluation of myocardial deformation and mechanical function by enabling the assessment of strain parameters with less angle dependency. Therefore, in the present study, the absence of STE-based strain analysis may have limited characterization of subtle changes in myocardial mechanics. Prospective studies with larger sample sizes and advanced echocardiographic techniques, including STE, are warranted to further validate and extend our findings. Third, serum digoxin concentrations were not measured; therefore, the relationship between therapeutic drug levels and improvement in mechanical synchrony could not be evaluated. Fourth, echocardiographic measurements were not analyzed in a blinded core laboratory, introducing the possibility of observer bias. Finally, indices of left ventricular filling pressure, such as the E/e' ratio, were not systematically incorporated into the analysis, although they may influence ventricular mechanical delay.

CONCLUSION

This study demonstrates that digoxin therapy is associated with significant improvements in mechanical synchrony, systolic function, ventricular remodeling, and electrical conduction in patients with non-ischemic HF. The observed reductions in intraventricular and interventricular dyssynchrony, combined with improvements in left ventricular performance and QRS duration, suggest that digoxin may exert beneficial effects on both mechanical and electrical aspects of cardiac function.

These findings support a broader therapeutic role for digoxin beyond symptom control and neurohormonal modulation, highlighting its potential as an adjunctive therapy aimed at improving mechanical efficiency and structural remodeling in HF. While the results are promising, larger, multicenter studies with longer follow-up and advanced imaging modalities are required to confirm these findings and to determine their impact on clinical outcomes such as hospitalization rates, functional capacity, and survival.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Local Ethics Committee of Erciyes University (decision no. 2016/381; date: 24 June 2016).

Informed Consent: All participants provided written informed consent in accordance with the principles of the Declaration of Helsinki.

Footnotes

Authorship Contributions

Surgical and Medical Practices: H.O., A.O.B., C.K., Concept: H.O., A.O.B., C.K., Design: H.O., A.O.B., C.K., Data Collection or Processing: H.O., A.O.B., C.K., Analysis or Interpretation: H.O., A.O.B., C.K., Literature Search: H.O., A.O.B., C.K., Writing: H.O., A.O.B., C.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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