



LEFT VENTRICULAR LAYER-SPECIFIC STRAIN BY 2D SPECKLE-TRACKING ECHOCARDIOGRAPHY IN PEDIATRIC MYOCARDITIS

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Abstract

Objective: Myocarditis is an inflammatory disease of the myocardium with variable clinical presentation and challenging diagnosis, particularly in children with preserved left ventricular ejection fraction. Layer-specific strain analysis using speckle-tracking echocardiography allows separate assessment of endocardial, mid-myocardial, and epicardial deformation. We aimed to evaluate layer-specific strain in pediatric patients with acute myocarditis to determine whether layer-specific deformation parameters detect subclinical myocardial dysfunction.

Methods: Two-dimensional and speckle-tracking echocardiography with layer-specific strain analysis were performed. Global longitudinal strain and layer-specific longitudinal strain were measured at basal, mid-ventricular, and apical levels in endocardial, mid-myocardial, and epicardial layers.

Results: Twenty-one pediatric patients with acute myocarditis (mean age 187.6 ± 22.3 months; 17 male) and 20 age- and sex-matched healthy controls were enrolled. Patients with myocarditis had lower left ventricular ejection fraction compared to controls ($63 \pm 3.4\%$ vs $71.2 \pm 1.9\%$, $p < 0.001$). Global longitudinal strain was reduced in the myocarditis group (-18.3 ± 2.9 vs -22.2 ± 1.3 $p = < 0.001$). All three myocardial layers showed reduced average longitudinal strain in patients compared to controls: endocardial ($-22.6 \pm 6.8\%$ vs $-25.7 \pm 1.7\%$, $p = 0.033$), mid-myocardial ($-18.1 \pm 2.9\%$ vs $-22.4 \pm 1.8\%$, $p = 0.040$) and epicardial ($-15.8 \pm 2.4\%$ vs $-20.2 \pm 1\%$, $p = 0.008$).

Conclusion: Layer-specific strain analysis detects subclinical myocardial dysfunction in pediatric myocarditis even when left ventricular ejection fraction is relatively preserved. Particularly epicardial strain impairment, reflects the known subepicardial predominance of inflammation in myocarditis. Layer-specific strain may provide incremental diagnostic value over conventional echocardiography in this population.

Keywords: Layer-Specific Strain, Pediatric, Myocarditis, Speckle tracking echocardiography.

Introduction

The diagnosis of acute myocarditis can be challenging due to the lack of specific diagnostic features. Presently, no single clinical or imaging finding can unequivocally confirm a diagnosis of myocarditis. Instead, diagnosis and treatment decisions should be guided by an integrated synthesis of clinical history, physical examination, and noninvasive test results.¹ In its most fundamental definition, myocarditis is characterized by inflammatory infiltration and subsequent destruction of cardiomyocytes. These pathologic changes predominantly affect the subepicardial and mid-myocardial layers, with relative sparing of the subendocardium—a pattern that distinguishes myocarditis from ischemic injury.² Myocarditis encompasses a broad spectrum of etiologies, predominantly of infectious origin, with viral pathogens representing the most frequently implicated agents. Beyond this, the condition arises from a diverse array of other infectious triggers as well as numerous noninfectious causes, including autoimmune disorders, hypersensitivity reactions, and exposure to specific pharmacologic agents or toxins. Myocarditis in the pediatric population presents multifaceted clinical challenges, encompassing the determination of an optimal diagnostic strategy, the calibration of therapeutic intervention intensity, and the design of appropriate long-term surveillance following the acute phase of illness. A significant proportion of patients experience spontaneous resolution, while a comparable proportion sustain irreversible myocardial injury. This dichotomy often necessitates clinical decisions—regarding both management and therapeutic intervention—in the absence of a confirmed diagnosis or robust evidence-based guidance. The diagnostic paradigm for myocarditis has evolved from a strict requirement for histopathological confirmation to a contemporary model that increasingly integrates non-invasive imaging, specifically cardiac magnetic resonance (CMR), with comprehensive clinical and laboratory assessment. The 2021 Scientific Statement from the American Heart Association delineates a four-tiered diagnostic framework for myocarditis, stratified by level of evidence: 1. biopsy-proven, 2. CMR-confirmed clinically suspected, 3. clinically suspected, and 4. possible myocarditis. This schema formalizes the contemporary shift from a purely histopathological definition to an integrated, multimodal diagnostic paradigm.³ The current reference standard for noninvasive diagnosis of myocarditis is CMR imaging according to the Lake Louise consensus criteria.¹ Two-dimensional speckle-tracking echocardiography (2DSTE) has advanced the capabilities of conventional echocardiography by enabling a quantitative assessment of both global and regional left ventricular (LV) function, thereby facilitating the detection of subclinical myocardial impairments. The advent of contemporary software further refines this analysis through layer-specific strain imaging, permitting the differentiation of endocardial and epicardial deformation.⁴ Consequently, these advanced imaging modalities may offer diagnostic advantages over conventional echocardiography in the evaluation and management of myocarditis.

This study sought to characterize alterations in myocardial layer-specific strain (LSS), quantified by 2DSTE, in patients with CMR imaging-confirmed myocarditis, and to evaluate the diagnostic utility of this metric within the current diagnostic algorithm. While CMR is the cornerstone imaging modality for tissue characterization, its access can be limited, particularly in pediatric populations where challenges such as the need for sedation, limited availability of child-appropriate protocols, and required specialist expertise constrain its routine use. The

investigation of LSS—which can differentiate dysfunction in the inner (endocardial), middle (mid-myocardial), and outer (epicardial) layers of the heart wall—proposes a more accessible echocardiography-based method that may detect subtle, early myocardial injury not visible on standard ultrasound.

Methods

The study was conducted on patients diagnosed with myocarditis at the Pediatric Cardiology Clinic of Kocaeli University between May 2021 and August 2025. Twenty age- and sex-matched healthy children were used as a control group and underwent two-dimensional echocardiography, speckle tracking, and layer-specific strain measurements. The diagnosis of acute myocarditis was established according to the integrated clinical (acute chest pain or acute dyspnea), laboratory (elevated troponin levels, abnormal electrocardiogram changes), and echocardiography imaging data set forth in the 2021 American Heart Association Scientific Statement, Diagnosis and Management of Myocarditis in Children.¹ CMR imaging was performed on the patients during their hospital admissions for treatment. Individuals with known rheumatologic disease, positive SARS-CoV-2 serology, a diagnosis of Multisystem Inflammatory Syndrome in Children (MIS-C), pre-existing cardiomyopathy, congenital heart disease, or inadequate echocardiographic image quality were excluded. For all participants, demographic characteristics and conventional echocardiographic indices, including left ventricular ejection fraction (EF) and fractional shortening (FS), were recorded. In addition to conventional measures, comprehensive myocardial deformation analysis was performed using Vivid E9 (GE) Software EchoPAC V113 (GE). Global longitudinal strain (GLS) was derived from apical four-chamber, two-chamber, and long-axis views using 2DSTE. Furthermore, a LSS analysis was conducted to quantitatively assess the contractile function of the endocardial, mid-myocardial, and epicardial layers separately within the basal, midventricular, and apical segments of the left ventricle. Peak systolic strain was defined as the maximum magnitude of either peak negative strain (indicating myocardial shortening) or peak positive strain (indicating myocardial lengthening) occurring during systole. Peak systolic longitudinal strain was evaluated in each of the 17 longitudinal LV segments. GLS was subsequently derived by averaging the segmental strain values.

Statistical analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Normally distributed variables are presented as mean $\pm$ standard deviation (SD); non-normally distributed variables are expressed as median (interquartile range [IQR] or range). Categorical variables are summarized as frequencies and percentages. Comparisons between the study and control groups were performed. For normally distributed continuous variables, the independent-samples Student's *t*-test was used. For non-normally distributed continuous variables, the Mann-Whitney *U* test was applied. Categorical variables (e.g., sex distribution) were compared using the Chi-square test or Fisher's exact test, as appropriate. Correlation analyses between continuous variables were conducted using Pearson's correlation coefficient when both variables were normally distributed, and Spearman's rank correlation coefficient when at least one variable deviated from normality. The specific test chosen is indicated in the results for each correlation. A *p* value < 0.05 was considered statistically significant. No adjustment

for multiple comparisons was applied given the exploratory nature of the study; results should be interpreted accordingly.

Results

The study included 21 patients with acute myocarditis (17 male, 4 female) and 20 healthy controls matched for age and sex (16 male, 4 female). The mean age of the myocarditis group was 187.6 ± 22.3 months (range : 144-214), and mean weight 66.3 ± 17.7 kg (Table 1).

Table 1. The demographic characteristics of the study group and control group.

	Study group (n=21)	Control group (n=20)	p value
Male /Female	17 /4	14/6	0.86
Age (months)	187.6 ± 22.3	186.1 ± 25.8	0.24
Weigh (kg)	66.3 ± 17.7	60.0 ± 11.8	0.073

All patients presented with chest pain and/or dyspnea with elevated high sensitive cardiac troponin I (hs-cTnI) level. Mean hs-cTnI level was 1262ng/L (range: 32- 8400). Seventeen patients had CRP values above 5 mg/L, and 8 patients had ESR values above 20 mm/hour. Baseline laboratory values are presented in Table 2.

Table 2. Baseline laboratory values of the study cohort.

	Mean±SD	Min. – Max.
Hs-cTnI (ng/L)	1262.4 ± 2141.6	32 - 8400
NT-proBNP (ng/L)	604.1 ± 548.5	70 - 1730
CK-MB (mcg/L)	36.3 ± 29.8	2 - 105
CRP (mg/L)	62.7 ± 72.9	1 -284
ESR (mm/hour)	20.8 ± 21.5	3 -88
WBC (/mm3)	8704 ± 3699.9	4020 -21210

Hs-cTnI: High-Sensitivity Cardiac TroponinI, *CK-MB*: Creatine kinase-myocardial band, *NT-proBNP*: N-terminal pro-B-type natriuretic peptide, *CRP*: C -reactive protein, *ESR*: Erythrocyte sedimentation rate, *WBC*:White blood cell count.

In the Spearman correlation analysis conducted within the study cohort, a strong negative correlation was identified between ejection fraction (EF) and C-reactive protein (CRP) ($r = -0.608$, $p = 0.003$), as well as a moderate negative correlation with erythrocyte sedimentation rate (ESR) ($r = -0.517$, $p = 0.023$). In contrast, no statistically significant correlations were observed between EF and troponin, CK-MB, or proBNP. A significant negative correlation was found only between CRP level and GLS ($r = -0.603$, $p = 0.022$). No significant relationship was observed between GLS and the cardiac biomarkers. A significant negative correlation was found between CRP level and layer specific longitudinal strain values obtained from three myocardial layers (endocardial: $r = -0.591$, $p = 0.020$; mid-myocardial: $r = -0.709$, $p = 0.003$; epicardial: $r = -0.586$, $p = 0.022$). In contrast, no significant relationship was observed between troponin, proBNP, or CK-MB and the myocardial layer-strain parameters. No statistically significant relationship was detected between the cardiac biomarkers and segmental longitudinal strain parameters. Negative but non-significant correlations were observed between CRP levels and apical ($r = -0.350$, $p = 0.169$), mid ($r = -0.128$, $p = 0.625$), and basal longitudinal strain values ($r = -0.179$, $p = 0.492$). On

baseline ECG, ST segment elevation was observed in 10 patients, whereas premature ventricular contraction was noted in only one patient. Mean hospital stay was 4.8 ± 1.2 days (range: 2-9). All patients received supportive therapy. No in-hospital mortality occurred. Conventional echocardiographic parameters and GLS are compared in Table 3. Patients with myocarditis had lower LVEF and FS compared to controls. GLS was significantly reduced in the myocarditis group (-18.3 ± 2.9 vs -22.2 ± 1.3 $p = <0.001$), indicating subclinical systolic dysfunction despite mean LVEF remaining within the normal range. The average longitudinal strain of the three (basal, mid-ventricular, and apical) segments exhibited a statistically significant reduction at the endocardial, mid-myocardial, and epicardial layers in patients with myocarditis compared to controls (Table 3).

Table 3. Comparison of transthoracic two-dimensional echocardiographic and speckle-tracking-derived parameters between the study population and healthy controls. EF: Ejection fraction, FS: Fractional shortening, GLS: Global longitudinal strain.

	Study group (n=21)	Control group (n=20)	p value
EF (%)	63.0 ± 3.4	71.2 ± 1.9	< 0.001
FS (%)	32.7 ± 1.9	35.9 ± 1.1	0.008
GLS (%)	-18.3 ± 2.9	-22.2 ± 1.3	< 0.001
Average Longitudinal Endocardial (%)	$-22.6 \pm 6.8 \%$	$-25.7 \pm 1.7 \%$	0.033
Average Longitudinal Mid-myocardial (%)	$-18.1 \pm 2.9 \%$	$-22.4 \pm 1.8 \%$	0.040
Average Longitudinal Epicardial (%)	$-15.8 \pm 2.4 \%$	$20.2 \pm 1 \%$	0.008

In both the study and control cohorts, endocardial GLS consistently showed the highest values, followed by mid-myocardial GLS, with epicardial GLS being the lowest at the base, mid-ventricle, and apex of the LV. Additionally, a longitudinal strain gradient from the apex (highest) toward the base (lowest) of the LV was observed across all three myocardial layers (endocardial, mid-myocardial, and epicardial). Consequently, strain gradients were present both transmurally from the endocardium to the epicardium and longitudinally from the apex toward the base of the LV (Table 4). Notably, apical endocardial strain was relatively preserved ($p = 0.121$), while epicardial strain showed significant impairment at all levels. The epicardial layer demonstrated the most consistent and marked impairment, with a reduction of approximately 14-25% compared to controls across different segments.

CMR imaging performed during the acute phase demonstrated late gadolinium enhancement (LGE) in 19 of 21 patients (90%). The LGE distribution was predominantly subepicardial and mid-myocardial (Figure 1), with involvement of the inferolateral and anterolateral walls in 16 patients (84%). This distribution pattern corresponded closely with the layer-specific strain abnormalities observed on echocardiography, particularly the marked reduction in epicardial strain in the basal and mid-myocardial segments (Figure 2).

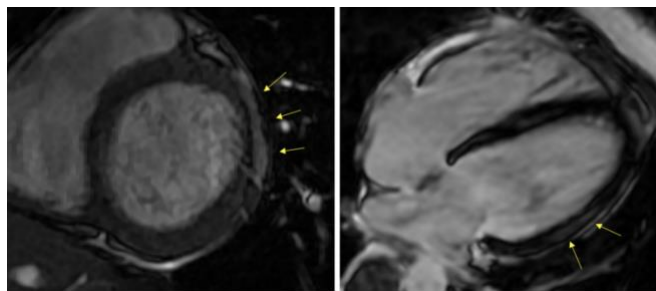


Figure 1. CMR short-axis and long-axis images showing epicardial and mid-myocardial (arrows) contrast enhancement of the left ventricular lateral wall in a patient with myocarditis.



Figure 2. Layer-specific strain values in the epicardial, mid-myocardial, and endocardial layers, demonstrating marked strain impairment in the epicardial layer, followed by the mid-myocardial and endocardial layers. In the left ventricle, strain impairment is more pronounced in the basal segment compared to the mid and apical segments.

Table 4. Comparison of left ventricular layer-specific longitudinal myocardial strain parameters at the basal, mid-ventricular, and apical levels between the study cohort and healthy controls.

Longitudinal Strain Segments	Study group (n=21)	Control group (n=20)	p value
Basal			
Endocardial (%)	-16.6 ± 2.6	-20.8 ± 1.1	< 0.001
Mid-Myocardial (%)	-15.6 ± 1.8	-20.2 ± 1.7	< 0.001
Epicardial (%)	-14.8 ± 2.6	-20.0 ± 0.8	< 0.001
Midventricular			
Endocardial (%)	-23.3 ± 2.1	-23.8 ± 1.3	0.6
Mid-Myocardial (%)	-18.2 ± 2.6	-21.9 ± 1.5	< 0.001
Epicardial (%)	-16.5 ± 2.3	-20.7 ± 1.2	< 0.001
Apical			
Endocardial (%)	-28.1 ± 5.7	-32.7 ± 2.9	< 0.001
Mid-Myocardial (%)	-20.6 ± 4.3	-25.3 ± 2.4	< 0.001
Epicardial (%)	-16.1 ± 2.5	-20.1 ± 1.2	< 0.001

Discussion

This study demonstrates that layer-specific strain analysis by 2DSTE detects subclinical left ventricular systolic dysfunction in pediatric patients with acute myocarditis, even when conventional LVEF is relatively preserved. Our findings show a consistent pattern of impairment across all three myocardial layers, with the epicardial layer being most severely and consistently affected. This pattern—epicardial > mid-myocardial > endocardial involvement—directly reflects the known pathophysiology of acute myocarditis, wherein inflammatory injury predominantly affects the subepicardial and mid-myocardial layers with relative sparing of the subendocardium.^{1,5} The robust negative correlation observed between CRP and layer-specific strain values, coupled with the absence of significant associations between EF, GLS, layer-specific strain and troponin, CK-MB, proBNP, indicates that in patients with myocarditis, left ventricular EF and GLS are significantly and inversely related to inflammatory

markers—particularly CRP—but not to conventional cardiac biomarkers. These findings suggest that impairment in ventricular systolic function and myocardial deformation may be more closely associated with systemic and/or local inflammatory processes than with the extent of myocardial injury reflected by troponin release. The pathophysiological basis for the layer-specific strain pattern observed in our study is well-established in the literature. Experimental and human histopathological studies have consistently demonstrated that viral myocarditis typically involves the subepicardial and mid-myocardial layers, with relative sparing of the subendocardium.⁶ This distinct pattern of involvement may serve as a non-invasive echocardiographic signature that helps differentiate myocarditis from other etiologies of myocardial dysfunction, particularly ischemic injury which typically shows subendocardial predominance.^{7,8} The transmural gradient of myocardial strain in healthy hearts, with highest values in the endocardium and progressive reduction toward the epicardium, has been well documented in both echocardiographic and CMR studies. This gradient reflects differences in wall stress, coronary perfusion, and myofiber architecture.^{9,10} The superior sensitivity of strain imaging, particularly epicardial strain, suggests that LSS analysis should be considered as an adjunctive diagnostic tool in patients with suspected myocarditis and preserved LVEF. Limited data exist on layer-specific strain in pediatric populations. Our study is among the first to apply echocardiographic LSS analysis to pediatric myocarditis and demonstrates that the layer-specific pattern of dysfunction mirrors that observed in adults.

In the pediatric population, the high quality of echocardiographic images and the ability of speckle tracking echocardiography to detect regional wall motion abnormalities render this modality particularly valuable for the assessment of pediatric myocarditis. Conversely, the use of CMR imaging in children remains suboptimal, owing to the need for sedation in young children, limited access to personnel with specialized expertise, higher costs, and patient noncompliance. The detection of subclinical myocardial dysfunction by LSS has several potential clinical applications in pediatric myocarditis. First, LSS may improve diagnostic confidence in cases where CMR is not readily available or contraindicated. While CMR remains the noninvasive gold standard, echocardiography is more widely accessible and can be performed at the bedside in critically ill children. Second, serial strain measurements may provide a noninvasive means of monitoring disease evolution and treatment response. The persistence of strain abnormalities despite clinical recovery could identify patients at risk for adverse remodeling or recurrent myocarditis. Third, the differential involvement of myocardial layers may help distinguish myocarditis from other causes of acute chest pain. Fourth, LSS may inform risk stratification and guide decisions regarding exercise restriction and return to sports participation—an important consideration in pediatric and adolescent patients.

Study Limitations

Our study had some limitations. First, the sample size was relatively small. Larger multicenter studies are needed to validate our findings and establish robust diagnostic cutoffs. Since the CMR imaging (performed during the treatment period) and the speckle-tracking echocardiographic assessment in our study were not conducted concurrently, the association between the findings derived from the two imaging modalities may lack concordance. Future studies should incorporate

simultaneous echocardiography and CMR to elucidate the relationship between layer-specific strain and myocardial inflammation. Our study population included predominantly adolescent patients with mild-to-moderate disease and relatively preserved LVEF; the generalizability of our findings to younger children or those with fulminant myocarditis requires further investigation. Due to inter-vendor variability in strain measurements, our results may not be directly comparable to those obtained with other ultrasound platforms.

Conclusion

Layer-specific strain analysis by two-dimensional speckle-tracking echocardiography detects subclinical left ventricular dysfunction in pediatric patients with acute myocarditis, even when conventional ejection fraction is relatively preserved. The pattern of involvement—with significant impairment in epicardial and mid-myocardial layers, and relative preservation of apical endocardial strain—reflects the known subepicardial predominance of inflammatory injury in this condition. LSS provides incremental diagnostic value over conventional echocardiography and may serve as a useful adjunct in the noninvasive assessment of pediatric myocarditis, particularly in settings where CMR is unavailable or deferred. Further studies are warranted to validate these findings and define the prognostic implications of layer-specific strain abnormalities in childhood myocarditis.

Ethics Approval

The study was approved by Kocaeli University Non-Interventional Research Ethics Committee. Approval number is KÜ GOKAEK-2026/04/46 and approval date is 12.02.2026. All patient data were handled in compliance with institutional and national data protection regulations. Identifying information was removed, and only anonymized data were used for analysis. Access to the data was restricted to authorized study personnel. Because this study was based on a retrospective review of existing medical records and did not involve direct patient contact, individual informed consent was waived by the institutional ethics committee.

Author Contributions

G.A., E.Z.B., K.B.: Concept ;Design; E.Z.B., K.B.: Supervision; Critical Review ;E.Z.B., A.M.Ç., Y.A.: Materials; G.A., E.Z.B., A.M.Ç., Y.A.: Data Collection and/or Processing; G.A., E.Z.B., Y.A., K.B.: Analysis and/or Interpretation; G.A., E.Z.B., A.M.Ç.: Literature Review; G.A., E.Z.B.: Writing; Resource – none;

Conflict of Interest

No conflict of interest or common interest has been declared by authors.

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