CARDIOLOGIE TUNISIENNE

ARTICLE ORIGINAL

Diagnostic Role of Cardiac Magnetic Resonance Imaging and speckle tracking echocardiography in MINOCA

Rôle diagnostique de l'imagerie par résonance magnétique cardiaque et de l'échocardiographie 2D strain dans les MINOCA

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Summary

Introduction: Acute myocardial injury with non-obstructive coronary arteries is a diverse but relatively rare clinical entity, often associated with diagnostic challenges. Determining the underlying etiology, whether ischemic or non-ischemic, is critical for appropriate management.

Objective: To evaluate the diagnostic contribution of cardiac imaging, particularly CMR and speckle-tracking echocardiography and investigate correlations between CMR findings and 2D strain echocardiography.

Methods: We conducted a prospective single-center study including patients admitted for acute chest pain, elevated troponin, and non-obstructive coronary arteries and underwent transthoracic echocardiography with 2D strain, and CMR. Strain values were analyzed globally and segmentally.

Results: The incidence of myocardial injury with non-obstructive coronaries was 3% among all acute coronary syndromes. The mean age was 47.3 ± 16.3 years, with male predominance and smoking as the leading risk factor (67.7%). Using a structured diagnostic algorithm, an etiological diagnosis was established in 90% of cases: myocarditis (38.7%), MINOCA (32.3%), and Takotsubo syndrome (19.4%). A CRP level >16 mg/L and a troponin peak >16 ng/mL were indicative of inflammatory and ischemic etiologies, respectively. A positive correlation was observed between troponin peak and both myocardial edema and LGE extent on CMR. While no segmental correlation was found between strain alterations and LGE distribution, GLS was significantly correlated with the overall extent of LGE. Both peak troponin and myocardial edema extent were predictive of impaired GLS recovery at follow-up.

Conclusion: 2D strain echocardiography is a useful and accessible tool for early evaluation, but CMR remains the gold standard for etiological diagnosis and patient management.

Keywords

Myocardial injury, MINOCA, myocarditis, cardiac magnetic resonance imaging, 2D strain echocardiography

Résumé

Introduction: La lésion myocardique aiguë avec artères coronaires non obstructives est une entité clinique rare mais hétérogène, souvent associée à des difficultés diagnostic. L'identification de l'étiologie sous-jacente, qu'elle soit ischémique ou non ischémique, est essentielle pour une prise en charge appropriée.

Objectif : Évaluer la contribution diagnostic de l'imagerie cardiaque, en particulier l'IRM cardiaque (CMR) et l'échocardiographie 2D strain, et explorer les corrélations potentielles entre les deux modalités.

Méthodes : Nous avons mené une étude prospective monocentrique incluant 31 patients admis pour douleur thoracique aiguë, élévation de troponine et coronaires non obstructives dans notre centre. Tous les patients ont bénéficié d'une évaluation clinique, de tests biologiques, d'une coronarographie, d'une échocardiographie transthoracique avec analyse strain 2D et d'une IRM cardiaque. Les valeurs de strain ont été analysées globalement et segmentairement.

Résultats : L'incidence des lésions myocardiques avec coronaires non obstructives était de 3 % parmi l'ensemble des syndromes coronariens aigus. L'âge moyen était de 47,3 ± 16,3 ans, avec une prédominance masculine et le tabagisme comme principal facteur de risque (67,7 %). Grâce à un algorithme diagnostic structuré, un diagnostic étiologique a été établi dans 90 % des cas : myocardite (38,7 %), MINOCA (32,3 %) et syndrome de Takotsubo (19,4 %). Aucune association significative n'a été retrouvée entre la présentation clinique initiale (STEMI/NSTEMI) et le diagnostic final. Un taux de CRP >16 mg/L et un pic de troponine >16 ng/mL étaient respectivement indicatifs d'étiologies inflammatoires et ischémiques. Une corrélation positive a été observée entre le pic de troponine et l'étendue de l'œdème myocardique ainsi que l'extension du rehaussement tardif (LGE) à l'IRM. Bien qu'aucune corrélation segmentaire n'ait été retrouvée entre les altérations du strain et la distribution du LGE, le strain longitudinal global (GLS) était significativement corrélé à l'étendue globale du LGE. Les valeurs de strain sous-endocardiques et sous-épicardiques n'ont pas montré de valeur diagnostic supplémentaire. Cependant, l'analyse segmentaire du strain a mis en évidence une atteinte de la paroi latérale comme possible indicateur de myocardite. Le pic de troponine et l'étendue de l'œdème myocardique étaient prédictifs d'une récupération altérée du GLS au suivi.

Conclusion : L'échocardiographie 2D strain est un outil diagnostique précieux, accessible et non invasif pour l'évaluation précoce des lésions myocardiques avec coronaires non obstructives. Bien qu'elle ne puisse remplacer l'IRM, elle apporte des informations cliniquement pertinentes, notamment lorsque l'IRM n'est pas disponible.

Mots-clés

Lésion myocardique, myocardite, syndrome de Takotsubo, imagerie par résonance magnétique cardiaque, échocardiographie 2D strain, strain longitudinal global, coronaires non obstructives

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INTRODUCTION

Acute coronary syndromes (ACS) is one of the principal causes of morbidity and death worldwide (1,2). While coronary angiography plays a central role in the diagnosis and management obstructive coronary artery disease (CAD), many patients with a diagnosis of myocardial infarction have no significant CAD on their coronary angiogram. This entity is known as myocardial infarction with non-obstructive coronary arteries (MINOCA), an entity that is described in 1–14% of cases of myocardial infarction patients (1). Described for the first time in 1939 (3), MINOCA is heterogeneous by its mechanisms and can have an embolic or atherosclerotic related origin making essential to accurately possess the underlying etiology and thus guiding the appropriate therapy and strategies for a better prognosis (4). Cardiac imaging plays a key role in the diagnostic work-up as it is essential to distinguish between different etiologies of MINOCA. Among the available modalities available for this purpose, cardiac magnetic resonance imaging (CMR) allows to tissue contrast so that a diagnosis of myocarditis, ischemic injury, Takotsubo syndrome, or other as well as the ischemic origin (5). MINOCA is still underreported and understudied in many parts of the world, including Tunisia, where there is limited information. Thus, we have conducted a single-center prospective study at the Hedi Chaker University Hospital in Sfax, Tunisia, aiming to describe the main features of patients with MINOCA, to address the diagnostic role of the different imaging modalities used including CMR to identify the enigma of the disease and the prognosis as well and explore the potential diagnostics of echocardiographic strain parameters, especially subendocardial and subepicardial strain, and findings of cardiac MRI.

METHODS

Study Design and Population

This was a prospective, single-center study conducted at the cardiology department of Hedi Chaker University Hospital in Sfax, Tunisia, over 19 months (June 2019 to December 2020). It included patients presenting with MINOCA.

Inclusion Criteria

Patients aged 15 to 85 years were included if they presented with a clinical picture consistent with acute myocardial infarction (MI) per the Fourth Universal Definition (6) (ESC 2018), associated with:

- Elevated troponin with a rising/falling pattern
- At least one of the following: ischemic symptoms, ECG changes, Q waves, imaging evidence of segmental wall motion abnormalities

AND coronary angiography showing either:

- Normal coronary arteries or
- Non-significant atherosclerotic plaque (<50%) without features of instability (e.g., thrombus, dissection)

Exclusion Criteria

Patients with prior coronary interventions, known myocarditis, heart failure, significant valvular disease, severe chronic kidney disease (CrCl <30 ml/min), pregnancy, acute non-cardiac conditions explaining troponin elevation (e.g., PE, sepsis), pacemakers, or contraindications to CMR were excluded.

Data Collection

Clinical, biological, electrocardiographic, and imaging data were systematically collected for all patients. 18-lead ECG was used to study rhythm, repolarization abnormalities, and the presence of Q waves. Laboratory investigations included a complete blood count, renal and liver function tests, C-reactive protein (CRP), troponin, and a coagulation profile. Transthoracic echocardiography was performed using a Philips EPIQ 7 echocardiograph, with evaluation of left ventricular ejection fraction (LVEF), wall motion score index (WMSI), valves evaluation, right ventricular function, and the presence or not of pericardial effusion. Myocardial deformation was analyzed using 2D speckle tracking imaging (STI) in the longitudinal plane, assessing global, subendocardial, and subepicardial longitudinal strain across all 17 myocardial segments.

Cardiac magnetic resonance imaging (CMR) was performed within 14 days of symptom onset using a 1.5 Tesla Siemens MRI scanner, or a Philips MRI scanner in some cases. The standardized imaging protocol included T2-weighted short tau inversion recovery (T2-STIR) sequences to assess myocardial edema, first-pass perfusion imaging to evaluate hyperemia, and late gadolinium enhancement (LGE) sequences to detect necrosis, inflammation, or fibrosis. Image interpretation was based on the Lake Louise criteria (2009) for the diagnosis of myocarditis (7) and the

American Heart Association (AHA) 17-segment model for tissue characterization (8). Based on CMR findings, patients were categorized into one of four groups: myocarditis, ischemic MINOCA, Takotsubo syndrome, or normal imaging findings.

DIAGNOSTIC STRATEGY AND DEFINITIONS

The diagnosis of MINOCA was established according to the 2018 ESC criteria (9), defined as myocardial infarction with non-obstructive coronary arteries in the presence of clinical and/or imaging evidence of ischemia. Myocarditis and Takotsubo syndrome diagnosis was based on the ESC guidelines and expert consensus (10), taking in consideration clinical data and CMR findings. A diagnostic algorithm was implemented, combining coronary angiography, transthoracic echocardiography—including strain imaging—and CMR. This multimodal assessment was performed within the first 48 hours to two weeks following symptom onset.

FOLLOW-UP

All patients were monitored in the coronary care unit for at least 24 hours. Clinical and echocardiographic follow-up (including GLS) was conducted at 1 months. Major adverse cardiac events (MACE) were defined as a composite of cardiac death, acute myocardial infarction, and rehospitalization for heart failure.

STATISTICAL ANALYSIS

Statistical analysis was conducted using SPSS version 20. Quantitative variables were expressed as means ± standard deviation, while qualitative variables were presented as counts and percentages. For comparisons, the chi^2 test or Fisher's exact test was used for categorical variables, and the Student's t-test or Mann–Whitney test was applied for continuous variables, depending on data distribution. Correlations were assessed using either Pearson or Spearman methods. Analysis of variance (ANOVA) was performed for comparisons involving more than two groups. Receiver operating characteristic (ROC) curve analysis was utilized to evaluate diagnostic performance. Finally, linear and multivariate regression analyses were carried out to identify independent predictive factors. A p-value of less than 0.05 was considered statistically significant.

RESULTS

During the 19-month study period, 1,032 coronary angiographies were performed for acute coronary syndrome (ACS), among which 31 patients (3%) had non-obstructive coronary arteries, meeting the criteria for myocardial injury with non-obstructive coronary arteries (MINOCA and mimickers).

The mean age was 47.3 ± 16.4 years (range: 16–81), with a predominance of males (81%). Smoking was the most prevalent cardiovascular risk factor (67.7%), followed by hypertension (19.4%) and diabetes (9.7%). Most patients (54%) had only one risk factor, while 22.6% had none.

16 patients presented with a STEMI and 15 with a NSTEMI. Angina-like chest pain was reported in 74.2%, anterior flu-like symptoms in 41.9% and emotional or physical stress triggers in 16%. 3 patients had hemodynamic instability.

ECG was abnormal in 83.9% of the cases with ST-segment elevation present in 54% of the patients, negative T waves in 25%, and Q waves in 19%. Mean troponin level was 13.7 ± 24.3 ng/mL, and CRP was 26 ± 40.2 mg/L.

Coronary angiography was performed via the radial approach in 96.8% of cases, revealing entirely normal findings in 54% of the patients. The other patients had minor, non-significant plaques on the angiogram. Echocardiography was conducted in the acute phase, with a median delay of 1-day after the hospitalization, showing a mean left ventricular ejection fraction (LVEF) of 55.3 ± 8.7%. Global longitudinal strain (GLS) was reduced (-14.4 ± 3%), with abnormalities affecting both subendocardial and subepicardial layers. The lateral wall had the most prominent impairment. CMR performed at a median of 2 days, was diagnostic in 82% of cases. Myocardial edema was identified in 13% (figure 1) , while late gadolinium enhancement (LGE) was found in 75% of patients—primarily subepicardial (figure 2)

(55%) or transmural (figure 3) (31%) in distribution. No-reflow areas were observed in two patients.

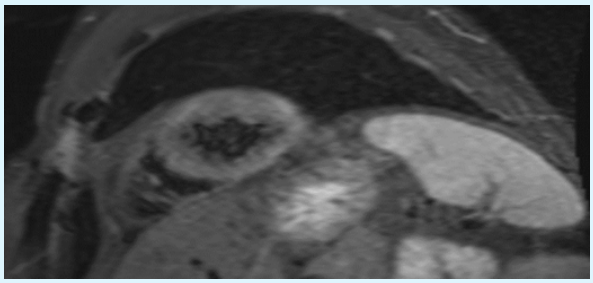

Figure 1. STIR sequence, short-axis view of the left ventricle: hypersignal of the lateral wall

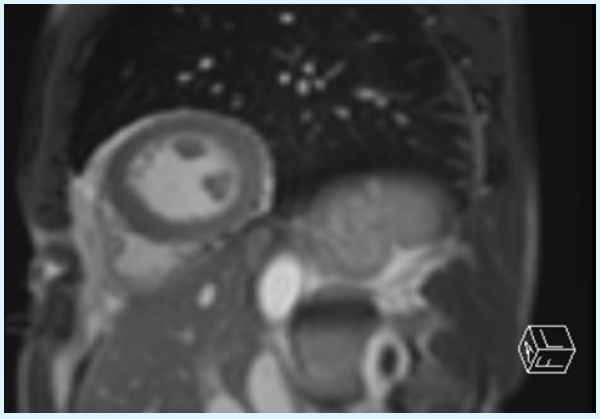

Figure 2. Late gadolinium enhancement (LGE) sequence, short-axis view: subepicardial LGE of the lateral wall

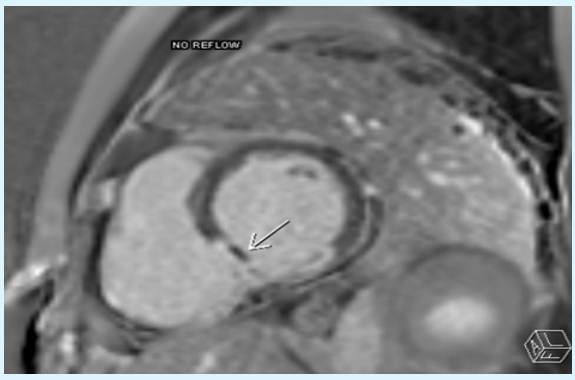

Figure 3. Late gadolinium enhancement (LGE) sequence, short-axis view: transmural LGE of the inferoseptal and inferior wall (red arrow) with no-reflow (white arrow)

The final diagnoses were distributed as follows: myocarditis was identified in 12 patients (39%), MINOCA of ischemic origin in 10 patients (32%), Takotsubo syndrome in 6 patients (19%), and the remaining 3 patients (10%) had unclassified etiologies.

All patients received standard ACS antithrombotic therapy initially according to the ESC guidelines. Etiology-specific treatment was later prescribed. Three patients experienced major adverse events (one death, one sustained ventricular tachycardia requiring ICD, and one MINOCA recurrence). Echocardiographic follow-up (mean = 3 months) showed recovery of LVEF and GLS in 83% of patients. The analytical component of this study revealed several key differences between patient groups as resumed in table 1. Notably, patients diagnosed with Takotsubo syndrome were significantly older, with a mean age of 64.7 years (p = 0.009), and showed a predominant female representation, contrasting with the male predominance in the myocarditis and MINOCA groups (p = 0.005). In terms of inflammatory and myocardial damage biomarkers, C-reactive protein (CRP) levels were substantially higher in myocarditis patients (36 mg/L vs. 4.7 mg/L; p = 0.007), while troponin levels were more elevated in the MINOCA group (21.5 ng/mL vs. 10 ng/mL; p = 0.035).

Table 1. Baseline Characteristics of Patients with Suspected Non-Obstructive Acute Myocardial Injury

Variable	Myocarditis (n=12)	MINOCA (n=10)	Takotsubo (n=6)	p-value
Male sex	100%	88%	33.3%	0.005
Age (years)	40.3 ± 17	47.1 ± 11	64.6 ± 13	0.009
Hypertension, n (%)	0 (0%)	1 (10%)	4 (67%)	0.002
Diabetes, n (%)	2 (16.7%)	1 (10%)	0 (0%)	0.651
Smoking, n (%)	10 (83.7%)	7 (70%)	2 (33.3%)	0.090
Obesity (BMI > 30 kg/m²), n (%)	4 (66.6%)	2 (20%)	0 (0%)	0.096
Flu-like syndrome, n (%)	7 (58%)	3 (30%)	1 (16.7%)	0.176
Major stress event, n (%)	0 (0%)	1 (10%)	3 (50%)	0.015
Anginal chest pain, n (%)	10 (83%)	9 (90%)	2 (33.3%)	0.104
STEMI clinical presentation, n (%)	7 (58.3%)	6 (60%)	2 (33.3%)	0.532
Systolic blood pressure (mmHg)	113 ± 16	117.8 ± 22	111.6 ± 24	0.803
Heart rate (bpm)	82.8 ± 32	82.1 ± 30	79.1 ± 17	0.972
Killip class IV, n (%)	1 (8.3%)	1 (10%)	1 (16.7%)	0.861
Normal ECG, n (%)	16%	20%	16%	—
ST-segment elevation, n (%)	66%	60%	33%	—
Negative T waves, n (%)	16%	20%	50%	—
Q waves (necrosis), n (%)	16%	30%	0%	—
QTc interval (ms)	420 ± 37	412 ± 17	435 ± 48	—
Ventricular arrhythmias, n (%)	16%	10%	0%	—

Receiver Operating Characteristic (ROC) analysis (figure 4) confirmed the diagnostic value of biomarkers: a troponin threshold of ≥4.4 ng/mL effectively predicted ischemic myocardial injury (AUC = 0.875), while a CRP threshold of ≥16 mg/L showed strong predictive ability for myocarditis (AUC = 0.844).

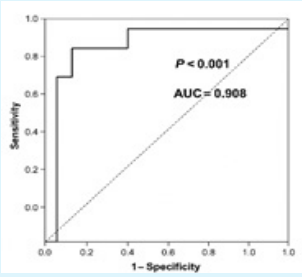

Figure 4. ROC Curve for CRP Value and Inflammatory Involvement

A significant association was revealed between troponin levels and the presence of myocardial edema (p = 0.03) as well as LGE (p = 0.019) by imaging analysis. However, no correlation was observed between troponin levels and the extent of these abnormalities. Additionally, troponin levels did not correlate significantly with GLS or WMSI.

Table 2. Left Ventricular Ejection Fraction and GLS Parameters in Patients vs. Healthy Controls

Parameter	Patient Group (n = 28)	Healthy Controls (n = 20)	p value
LVEF (%)	55 ± 8.7	69 ± 12	< 0.001
Mean GLS (%)	-14.42 ± 2.97	-18.78 ± 2	< 0.001
Subendocardial GLS (%)	-15.97 ± 3.38	-20.84 ± 2.2	< 0.001
Subepicardial GLS (%)	-12.87 ± 2.56	-16.55 ± 1.8	< 0.001
Mean GLS – Anterior Wall (%)	-14.67 ± 3.03	-18.04 ± 3.3	0.002
Mean GLS – Lateral Wall (%)	-13.75 ± 4.34	-18.88 ± 2.1	< 0.001
Mean GLS – Inferior Wall (%)	-15.41 ± 2.84	-20.3 ± 2.2	< 0.001
Mean GLS – Apical Wall (%)	-13.39 ± 5.51	-18.73 ± 2.3	< 0.001

Importantly, strain parameters (GLS, subendocardial, and subepicardial) were all significantly reduced in patients compared to healthy controls (p < 0.001) as showed in table 2. Notably, 25% of patients showed abnormal GLS despite preserved LVEF. A strong correlation was found between LVEF and GLS (p < 0.001) and between echocardiographic and CMR-derived LVEF (p = 0.027) as shown in Figure 5.

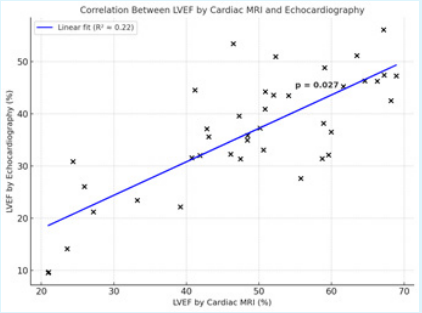

Figure 5. Late gadolinium enhancement (LGE) sequence, short-axis view: transmural LGE of the inferoseptal and inferior wall (red arrow) with no-reflow (white arrow)

Segmental analysis identified distinct regional patterns: myocarditis cases most often involved the lateral wall, while MINOCA cases showed predominant inferoseptal and inferior wall involvement. Although no statistically significant correlation emerged between segmental strain and LGE across segments, a trend was noted in segment 14 (p = 0.020).

Finally, multivariate regression (Table 3) identified two independent predictors of impaired myocardial strain recovery at follow-up: a higher troponin peak (p = 0.05) and a greater myocardial edema on CMR (p = 0.01).

Table 3. Predictive Factors of Poor Recovery of Mean GLS Assessed by ΔGLS, in Univariate and Multivariate Linear Regression Analysis

Variable	Univariate Analysis Multivariate Analysis		p value
	P-value	r	P-value
Age (years)	0.118	0.321	—
Creatinine (µmol/L)	0.387	0.683	—
QT interval (ms)	0.743	0.069	—
RT extension (sec.)	0.939	0.566	—
CPK (mg/L)	0.010	0.683	0.351
Peak troponin (ng/mL)	<0.001	0.683	0.050
Extent of myocardial edema (T2-STIR, segments)	0.004	0.601	0.010

DISCUSSION

Acute myocardial injury with non-obstructive coronary arteries is a challenging entity that lies at the intersection of ischemic and non-ischemic etiologies. In our study, this condition represented 3% of acute coronary syndrome (ACS) presentations, a proportion that aligns with previously reported

incidence rates ranging from 1% to 6% in large observational registries and contemporary clinical studies (11–13). Despite its relatively low incidence, its impact is great given the diagnostic ambiguity and therapeutic uncertainty that often surround these patients before finding the etiology.

Our work adds to existing literature by confirming that troponin remains an exceptionally sensitive marker for heart muscle damage; however, an elevated value still falls short of telling clinicians whether the injury was caused by reduced blood flow or by some other process. We found that people with ischemic damage (MINOCA) had higher troponin levels, whereas those with inflammatory etiology such as myocarditis showed much higher CRP levels. This matches earlier work stating that, while neither marker is foolproof alone, CRP and troponin interpreted together can steer diagnosis in the right direction (3,14,15). In fact, we found that a CRP level above 16 mg/L was associated with myocarditis with both high sensitivity and specificity, whereas a troponin cut-off of 16 ng/mL pointed more strongly toward MINOCA.

Coronary angiography remains the GOLD standard when there is a suspicion of an acute coronary syndrome, yet it almost never explains what caused the damage in this subgroup of patients. In our study, even the non-significant plaques seen on angiograms were not associated statistically to MINOCA ($p = 0.378$), joining past publications and reports concluding that up to half of MINOCA cases have atherosclerotic plaques nonvisible on regular coronary angiography and discovered only by intravascular imaging modalities (16,17). The findings show that conventional X-ray imaging is not enough and show how much we need to rely on modern imaging to establish the right diagnosis.

Although echocardiography is crucial during an emergency, in our cases it added little help for figuring out the etiology. Wall motion abnormalities and apical ballooning were observed in selected patients; however, these findings were not specific. The use of speckle-tracking echocardiography, and particularly the analysis of GLS, enhanced our ability to diagnose infraclinical myocardial dysfunction. In 25% of patients with preserved ejection fraction, GLS was impaired on admission showing its value as an early and sensitive marker of myocardial dysfunction (18–20). This finding is particularly relevant in settings where CMR is not available, which is the case in many hospitals in developing countries.

A unique feature of our work was the focus on subendocardial and subepicardial global strain (GLS) trying to link these measures with the underlying heart damage we observed on CMR since the subendocardial and subepicardial abnormalities are the key diagnostic features to differentiate between ischemic and non-ischemic etiologies. Earlier reports showed that myocarditis targets mainly the outer layer, whereas ischemia hits the inner layer (21–25), yet we saw no significant association between strain in either zone and CMR scars ($p = 0.964$ and 0.982, respectively). This lack of correlation may be secondary to the way the heart wall moves as a whole, rather than as separate segments. Because fibers twist helically and tissue cannot be compressed, strain in one layer inevitably spills over into adjacent layers-even those that appear normal on a microscope-and thus reduces the power of layer-specific readings (26–28). Still, our data add to a growing work suggesting that GLS changes speak to global muscle weakness instead of pinpointing a precise transmural injury site.

The drop in average GLS tracked closely with the area of late gadolinium enhancement seen on CMR, hinting that strain may act as an easy, bedside gauge of how much heart tissue has been harmed (29,30). Segmental strain patterns, however, rarely lined up perfectly with the LGE map yet the overall tie still paints GLS as a useful, non-invasive marker for myocardial damage.

In our workflow, CMR stood out as the best imaging modality, settling the diagnosis in 82% of patients, a rate that matches what centers in Germany and Australia have reported (31). The average three-day lag between the first symptom and the scan certainly helped, allowing us to catch fleeting edema and early inflammation before they faded away. T2-weighted and fat-suppressed STIR images highlighted the edema, whereas the pattern of LGE, whether subendocardial, transmural, mid-

wall, or subepicardial, helped us sort ischemic from non-ischemic lesions. Curiously, how much LGE there was failed to predict later improvement in heart function, a finding that backs recent papers challenging the importance of LGE volume in MINOCA and myocarditis patients (32).

When we looked at strain segment by segment, the lateral wall showed more injury in myocarditis, matching what other pediatric and adult published cohorts have reported about where fibrosis and weakness usually are found (30,33). Even though that trend fell short of statistical significance in our small sample, it probably makes sense to chase it in bigger studies. For MINOCA patients, we found that standard tests: ECG, troponin, even clean coronary pictures-still left the mechanistic puzzle unsolved. Because our center rarely runs intravascular ultrasound or optical coherence tomography and does not provoke spasm, we could not tell whether plaque rupture, a tight spasm, or an embolus was really at fault (30,34). We therefore treated everyone according to guidelines, starting statins, RAAS blockers, and antiplatelet therapy sensible yet second-best solution since experts say therapy should match the precise cause when it appears (35,36). Taken together, our study adds to the growing literature that patients with acute myocardial injury and non-obstructive coronary arteries are a mixed group and demand a broad, layered diagnostic algorithm.

CONCLUSION

Acute myocardial injury with non-obstructive coronary arteries includes a large spectrum of etiologies that cannot be differentiated by conventional diagnostic tools. Our study highlights the critical role of CMR in determining the etiology, with a diagnostic yield of over 80% when performed early during hospitalization. CMR was able to differentiate between ischemic and inflammatory patterns based on late gadolinium enhancement and tissue characterization sequences.

Speckle-tracking echocardiography, and particularly GLS, emerged as a sensitive marker of myocardial damage even in patients with preserved LVEF. Although no correlation was found between layer-specific strain and the distribution of injury on CMR, GLS was significantly associated with the extent of CMR abnormalities which suggests its utility as a non-invasive marker for injury and possibly for prognosis.

Our findings support the application of a stepwise, multimodal diagnostic algorithm for patients presenting with elevated troponin and non-obstructive coronaries. In resource-limited settings where CMR or intracoronary imaging are not readily available, GLS can offer valuable initial insights. Moreover, we identified prognostic indicators such as peak troponin and myocardial edema extent cut-offs may help stratify risk and guide follow-up.

Future studies with larger cohorts and integration of advanced imaging tools including CMR feature tracking, T1/T2 mapping, and intracoronary imaging are necessary to outline pathophysiological mechanisms, improving diagnostic accuracy in this heterogeneous patient population.

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