Impact of microvascular obstruction on left atrial and ventricular myocardial deformation and in-hospital complications in reperfused STEMI patients

Abstract

Objective: The purpose of this study was to explore the impact of microvascular obstruction (MVO) on left atrial and ventricular parameters, as well as in-hospital complications, in patients with reperfused ST-segment elevation myocardial infarction (STEMI) who underwent cardiovascular magnetic resonance (CMR) within 7 days.

Method: This retrospective study included CMR scans of 95 consecutive patients with reperfused STEMI (79 males, mean age 64.2 ± 10.3 years). Among them, 30 showed MVO presence (28 males, 93%; mean age 64.1 ± 10.5 years), defined as a hypo-enhanced core surrounded by hyper-enhanced myocardium in late gadolinium enhancement (LGE) sequences.

Results: Patients with MVO demonstrated a higher prevalence of hypertension ($p = 0.012$) and a lower LV ejection fraction (30.5 ± 9.1% vs. 37.8 ± 13.8%, $p = 0.011$) compared to patients without MVO. Additionally, patients with MVO showed impaired reservoir, conduit, booster function, global longitudinal strain (GLS), global circumferential strain (GCS), global radial strain (GRS), higher LGE extent, and in-hospital complications compared to STEMI patients without MVO ($p = 0.001$ for all).

In multivariable analysis, reservoir, conduit, booster function, GLS, GCS, and GRS were associated with MVO presence, independently of LV ejection fraction and LGE extent ($p = 0.001$ for all).

Conclusion: In reperfused STEMI patients, left atrial and ventricular strain parameters are altered by the presence of MVO, in addition to impaired LV ejection fraction and LGE extent.

Abbreviations

CMR cardiovascular magnetic resonance

GCS global circumferential strain

GLS global longitudinal strain

GRS global radial strain

LGE late gadolinium enhancement

LV left ventricle

MVO microvascular obstruction

STEMI ST-segment elevation myocardial infarction

Keywords: STEMI; myocardial strain; CMR.

Introduction

Microvascular obstruction (MVO) is a key phenomenon that frequently occurs following reperfused ST-segment elevation myocardial infarction (STEMI) and is strongly linked to adverse clinical outcomes, including left ventricular (LV) remodeling, heart failure, and increased mortality[1–4]. Despite successful transmural reperfusion, MVO—also known as the "no-reflow" phenomenon—represents the failure of microvascular blood flow, resulting in tissue damage that extends beyond the infarcted area[1]. Early detection of MVO is crucial as it serves as a significant prognostic marker and helps guide therapeutic interventions aimed at reducing long-term myocardial injury[1,3]. Cardiac magnetic resonance (CMR) imaging has become the gold standard for assessing MVO, offering comprehensive insights into myocardial viability, infarct size, ischemia, LV remodeling, and systolic function[5,6]. MVO can be evaluated through CMR using gadolinium contrast, which enables

assessment via two main methods: first-pass perfusion imaging (early MVO) and late gadolinium enhancement (late MVO)[1,2]. Studies have shown that, compared to early MVO, the presence and extent of late MVO are more robust independent predictors of long-term outcomes, including LV dysfunction and poor recovery[7]. Therefore, identifying the determinants of MVO and delineating the possible underlying mechanisms related to adverse outcomes in STEMI patients may help to better stratify patients after myocardial infarction.

Additionally, CMR-based feature-tracking techniques enable the evaluation of myocardial strain parameters, providing detailed insights into both global and regional myocardial function[8–14]. Strain imaging, which includes global longitudinal strain (GLS), global circumferential strain (GCS), global radial strain (GRS), and atrial strain parameters—such as reservoir, conduit, and booster strain—has demonstrated potential in detecting subclinical myocardial dysfunction in STEMI patients[15–20]. Unlike LV ejection fraction, which reflects only global systolic function and does not accurately capture regional functional abnormalities or diastolic dysfunction, strain imaging provides additional insights into myocardial mechanics [15–20].

However, the role of left atrial and ventricular strain parameters in the presence of MVO and their relationship with conventional markers such as LV ejection fraction and myocardial infarction extent remains underexplored. Gaining insight into these relationships could enhance the predictive value of CMR imaging and improve risk stratification in post-STEMI patients. This study aims at investigating the association between left atrial and ventricular strain, as well as in-hospital complications, and the presence of MVO in patients with reperfused STEMI during the acute/subacute phase.

Material and Method

Study population

In this retrospective, longitudinal, observational, single-center study, all consecutive patients with acute STEMI treated with PCI who underwent CMR within 7 days between March 3rd, 2013, and December 31th,2023 were included.

A STEMI diagnosis was established when chest pain persisted for at least 30 minutes, occurred within 12 hours of symptom onset, and was accompanied by an ST-segment elevation greater than 2 mm (0.2 mV) in at least two adjacent leads on the electrocardiogram

Exclusion criteria included: subjects < 18 years; previous myocardial infarction; pre-existing cardiomyopathy; and suspected or known prior irreversible myocardial damage.

Cardiovascular risk factors were collected from medical records. Hypertension was defined as a systolic blood pressure of ≥140 mmHg or a diastolic blood pressure of ≥90 mmHg at rest on more than two occasions, or the use of antihypertensive drugs[21]. Smoking status was defined as current smokers or never smokers. Cholesterol laboratory analyses were conducted following the standard in-house protocol. Diabetes status was assessed using the World Health Organization criteria[22] or an established diagnosis of type 2 diabetes. Obesity was defined as a BMI > 30, as defined by the World Health Organization criteria[23].

All hospital records were reviewed for clinical events during the patients' hospitalization. The primary endpoint was a composite of adverse cardiac events, including cardiovascular death, nonfatal reinfarction, heart failure, ventricular arrhythmia, and ischemic stroke. Recurrent acute myocardial infarction was diagnosed according to current guidelines of the European Society of Cardiology[24]. Stroke was defined as an ischemic cerebral infarction resulting from embolic or thrombotic occlusion of a major intracranial artery. Heart failure was assessed and classified based on the latest guidelines of the European Society of Cardiology[25]. Ventricular arrhythmia was defined as the presence of nonsustained ventricular tachycardia, sustained ventricular tachycardia, or aborted sudden cardiac death.

The Institutional Review Board approval for this retrospective, cross-sectional study was obtained, and patient's consent was waived because of the retrospective nature.

CMR acquisition

CMR scans were performed at 4.2±2.8 days (median = 1 day, range = 1-7 days) after admission to the hospital by using a Philips Achieva dStream 1.5 T scanner system (*Philips Healthcare, Best, The Netherlands*). Anterior coil arrays were used. All cine-images were acquired using a balanced steady-state free precession and retrospective gating during an expiratory breath- hold manoeuvres (TE: 1.7mssec; TR: 3.4msec /flip-angle: 45°, section thickness = 8 mm) in both long-axis (two-, three- and four-chamber view) as well as short-axis plane with whole ventricular coverage from base to apex. Ten to fifteen short-axis views covering the whole Left and Right Ventricle (LV and RV, respectively) were obtained.

LGE imaging was performed 10-12 minutes after contrast media injection (Gadovist, Bayer Healthcare, Berlin, Germany) with a dose of 0.15 ml per kg body weight using phase-sensitive inversion recovery sequences acquired in both short and long axis. The correct inversion time was determined using the Look-Locker technique.

CMR image post-processing

We used the commercially available software system Circle CVI42 (*CVI42, Circle Cardiovascular Imaging Inc., Calgary, Canada*) for CMR-FT data analysis. LV volumes and function were determined on short-axis cine images using an automatic computation to outline the endocardial and epicardial borders throughout the cardiac cycle. The quality of tracking and contouring was visually validated, manually corrected as needed, and consistently applied to all

patients. LV end-diastolic volume and end-systolic volume were obtained from the cine images. The LV ejection fraction (EF) was calculated using the formula: (LV end-diastolic volume – LV end-systolic volume) / LV end-diastolic volume * 100%.

Offline CMR feature tracking analyses were performed to evaluate peak global longitudinal strain, global radial strain, and global circumferential strain using a 16-segment, software-generated 2D model. Longitudinal strain data were derived from two-, three-, and four-chamber long-axis views, while radial and circumferential strain data were obtained from apical, mid-ventricular, and basal short-axis views in all patients. The epi- and endocardial borders were traced in end-diastole on all images, after which an automatic computation was initiated to outline the borders throughout the cardiac cycle. The quality of tracking and contouring was visually validated and manually corrected as needed.

CMR feature tracking analyses of atrial deformation were also conducted offline. The LA endocardial borders were manually traced on long-axis views of the cine images when the atrium was at its minimum volume. Specifically, the four-, three-, and two-chamber views were used to derive LA longitudinal strain, excluding the LA appendage and pulmonary veins. After manual segmentation, the software automatically tracked the myocardial borders throughout the entire cardiac cycle. The quality of tracking and contouring was visually validated and manually corrected by a radiologist with three years of experience in cardiac imaging. The strain curve included three peaks: reservoir, conduit, and booster strain.

MVO was identified visually on LGE images as a hypo-enhanced core surrounded by hyper-enhanced myocardium. An example of MVO is shown in **Figure 1.**

The LGE extent was evaluated both semiquantitatively and quantitatively. The semiquantitative assessment was based on the number of segments with an ischemic LGE pattern, according to the American Heart Association (AHA) 17-segment model[26,27]. The quantitative assessment was performed by tracing the epicardial and endocardial contours on each short-axis image. A region of interest was manually placed in the myocardium without LGE, using a signal

intensity threshold of 5 standard deviations above the normal remote myocardium, and expressed as a percentage of LV mass.[28]

Statistical analysis

Continuous variables are presented as mean ± standard deviation. Comparisons of continuous data were performed using the independent samples t-test or Mann-Whitney U test; Kolmogorov-Smirnov tests were used to check continuous variables for normal distribution. Categorical variables were compared by using the chi-square test or Fisher's exact test, as appropriate. Correlation was assessed using the Pearson r and Spearman rho coefficient as appropriate. Association between MVO, clinical and CMR parameters, and myocardial infarction were analyzed using univariable and multivariable linear regression.

A p-value <0.05 was considered statistically significant. All statistical analysis was performed using IBM SPSS Statistics version 22 (SPSS Inc., Chicago, IL, USA).

Results

Baseline characteristics

A total of 95 patients with reperfused STEMI, including 79 males (83%) and 16 females (17%) with a mean age of 64.2 ± 10.3 years, were enrolled in the study. The patients were divided into two distinct groups based on the presence of MVO. One group consisted of 30 patients with MVO (28 males, 93%; mean age 64.1 ± 10.5 years), while the other group consisted of patients without MVO (51 males, 78%; mean age 64.3 ± 9.3 years). No significant differences were observed between patients with and without MVO in terms of age and sex. Patients with MVO demonstrated a higher prevalence of hypertension (p = 0.012) compared to those without MVO. No other differences were observed in terms of cardiovascular risk factors.

During a median hospitalization of 5 days (IQR 2–18), 30 patients (32%) experienced adverse cardiac events, including 2 cases of cardiovascular death, 2 cases of nonfatal reinfarction, 16 cases of rehospitalization for heart failure, 6 cases of ventricular arrhythmia, and 4 cases of ischemic stroke. A total of 65 patients (58%) completed the hospitalization period without any events. Finally, patients with MVO demonstrated higher in-hospital complications in comparison with STEMI patients without MVO (56% vs 26%, p = 0.001).

Table 1 displays the comparison between the groups enrolled

Clinical parameters, cardiac biomarkers, and CMR features in STEMI patients.

CMR characteristics of the enrolled patients are summarized in Table 2. STEMI patients with MVO showed a lower LV ejection fraction (30.5 ± 9.1% vs. 37.8 ± 13.8%, p = 0.011). No other significant differences were observed in LV volumes, right ventricular volumes, or function. Patients with MVO demonstrated lower reservoir, conduit, and booster strain parameters (10.3 ± 6.1% vs. 19.6 ± 10.5%, p = 0.001; 4.4 ± 3.3% vs. 9.7 ± 8.4%, p = 0.001; and 6.1 ± 4.4% vs. 10.1 ± 5.5%, p = 0.001, respectively).

Regarding ventricular strain parameters, GLS, GCS, and GRS were more impaired in STEMI patients with MVO compared to those without MVO (-5 ± 1.5% vs. -9.1 ± 4.8%, p = 0.001; -6 ± 2.6% vs. -10.3 ± 4.6%, p = 0.001; and 9.3 ± 3.8% vs. 15.7 ± 9.1%, p = 0.001, respectively). Additionally, a more extensive myocardial LGE was observed in STEMI patients with MVO, with a mean myocardial segment involvement of 7.06 ± 2.3 segments compared to 5.04 ± 1.5 segments in patients without MVO (p = 0.001), and an LGE percentage of 29.26% ± 11.44 compared to 20.18% ± 10.23 in patients without MVO (p = 0.001).

Determinant of MVO in reperfused STEMI patients

Univariable and multivariable determinants of MVO are presented in **Table 3** and **Table 4**. Univariable analysis revealed that hypertension (β coefficient = -1.122, p = 0.014), LV ejection

fraction (β coefficient = -2.470, p = 0.014), reservoir (β coefficient = -3.676, p = 0.001), conduit (β coefficient = -3.205, p = 0.001), booster (β coefficient = -3.081, p = 0.002), GLS (β coefficient = 3.443, p = 0.001), GCS β coefficient = 3.807, p = 0.001), GRS (β coefficient = - 3.238, p = 0.001) as well as the number of myocardial segments involved by LGE (β coefficient = 3.311, p = 0.001) and LGE % (β coefficient = 3.354, p = 0.001) were associated with MVO.

Further multivariable analysis revealed that reservoir, conduit, booster, GLS, GCS, and GRS were independently associated with MVO presence, beyond LV ejection fraction and LGE extent (β coefficient = -3.935, p = 0.001; β coefficient = -3.412, p = 0.001; β coefficient = -3.414, p = 0.001; β coefficient = 3.689, p = 0.001; β coefficient = 3.906, p = 0.001; β coefficient = -3.381, p = 0.001, respectively).

Discussion

The results of this study highlight the significant impact of MVO on myocardial function in patients with reperfused STEMI during the acute/subacute phase. Using CMR imaging, we found that patients with MVO exhibited more pronounced impairment in both left atrial and ventricular strain parameters compared to those without MVO, as well as higher in-hospital complications. **Figure 2**. Additionally, left atrial and ventricular strain parameters were independently associated with the presence of MVO, beyond the extent of myocardial infarction and impaired LV ejection fraction.

Timely reperfusion in patients with myocardial infarction rescue myocardium from loss and reduces mortality, although unfortunately successful restoration of epicardial coronary artery patency after prolonged occlusion does not always result in adequate microvascular reperfusion[1]. MVO is influenced by multiple factors, including cytotoxic agents, local inflammation, vasoconstriction, distal microembolization of atherosclerotic debris, platelet plugs, and cellular edema[1]. Accurate detection and quantification of MVO is crucial, with some studies showing that it is independently associated with adverse ventricular remodeling and worsened patient prognosis[3,16–18]. CMR is an

essential diagnostic tool for assessing myocardial damage in reperfused myocardial infarction, particularly for identifying MVO. Delayed T1-weighted imaging after the administration of gadolinium-based contrast agents provides excellent visualization of infarcted tissue and can detect persistent LGE[4]. However, it is widely recognized that late MVO is less sensitive than early MVO because small no-reflow zones rapidly enhance due to contrast diffusion from surrounding areas with intact microvasculature. While late MVO may be more specific, as it reflects regions of true no-reflow, early MVO represents areas with minimal residual blood flow, suggesting that late MVO indicates more severely compromised microcirculation[7]. Considering the connection between MVO and adverse outcomes in STEMI patients after reperfusion, identifying factors associated with MVO could aid in risk stratification for these patients.

Patients with MVO demonstrated a more impaired LV ejection fraction. These findings are in line with the literature[29–31]. In the systematic review and meta-analysis by Hamirani et al. evaluating the impact of MVO on cardiac function and volumes, it was reported that 'late' MVO was associated with a more impaired LV ejection fraction (Inverse Variance -5.82, Confidence Interval −8.21 to −3.43)[30].

In line with previous studies, we have demonstrated that STEMI patients with MVO showed a higher infarct size. Malek et al. demonstrated that the presence of MVO, independent of the extent, corresponds to a larger infarct size [31]. Due to their larger size, MVO infarcts exert a more pronounced effect on global function and regional contractility, impacting not only the infarcted area but also the surrounding peri-infarct region. In fact, we found that STEMI patients with MVO after reperfusion exhibited impaired GLS, GCS, and GRS compared to those without MVO. However, in our multivariable logistic analysis, the impairment in all three myocardial layers remained significant even after adjusting for the extent of myocardial infarction, suggesting a multifactorial etiology of the peri-infarct region dysfunction. Paradoxically, reperfusion itself can cause additional myocardial damage, extending beyond the infarcted area. In the surrounding myocardium, altered loading conditions, mechanical tethering of viable tissue to necrotic regions,

and pro-inflammatory mechanisms are likely contributors, which may explain why dysfunction worsens in the presence of MVO[32]. Another hypothesis suggests that reduced elasticity in infarcted myocardium with extensive MVO leads to increased local wall stress in the surrounding healthy myocardium. This, in turn, results in greater LV stiffening and enlargement compared to infarcted tissue with open microvasculature[33]. Additionally, stunned myocardium in the adjacent region may contribute to early dysfunction following infarction[32].

Recent data indicate that sustained microvascular vasoconstriction following PCI, attributed to elevated neuropeptide-Y levels in coronary circulation, may lead to increased myocardial damage and dysfunction after reperfused STEMI[34].

The other interesting finding in our study was the detection of a more impaired left atrial function in STEMI patients with MVO. Given the impairment of LV performance, impaired left atrial function is expected due to the anatomical connection between the ventricle and the atrium. In patients with LV dysfunction, left atrial contraction increases to maintain optimal LV filling. Initially, this results in enhanced left atrial pump function, but also increases left atrial stiffness, leading to a mismatch between workload and function. As LV dysfunction progresses and afterload on the left atrial increases, pump function eventually declines[11,35–37].

Finally, STEMI patients with MVO demonstrated higher in-hospital complications compared to patients without MVO. These results are in line with the current literature, which shows worse outcomes in the presence of MVO[1,38–40].

This can be attributed to several interrelated mechanisms. First, MVO represents a failure of reperfusion at the microvascular level despite the restoration of epicardial coronary blood flow. Persistent microvascular obstruction leads to impaired myocardial perfusion, resulting in prolonged ischemia in the affected myocardial tissue and, consequently, greater myocardial damage[1]. Additionally, MVO has been shown to contribute to increased interstitial myocardial edema and prolonged inflammation, which further compromise myocardial function[1].

Clinical implications

The findings of this study emphasize the importance of identifying MVO in patients with reperfused STEMI during the acute/subacute phase following PCI, as it is associated with more extensive myocardial damage, higher in-hospital complications, and worse contractility across all three myocardial layers, as well as impaired atrial function. CMR-based strain imaging, which assesses both left atrial and ventricular function, provides additional insight into myocardial dysfunction beyond conventional markers such as LV ejection fraction and LGE extent. Incorporating strain analysis into the routine assessment of STEMI patients during this early phase could improve risk stratification and guide therapeutic decisions, potentially leading to better outcomes.

Limitations

This study has several limitations that should be noted. Firstly, the relatively small sample size restricts the generalizability of our findings. While our results are promising, larger studies are needed to confirm these findings. Secondly, the cross-sectional design of the study, along with the focus on the acute/subacute phase post-PCI, limits our ability to evaluate the predictive value of MVO for adverse cardiovascular events in STEMI patients and its impact on long-term LV remodeling. Third, MVO was assessed as a binary parameter, without considering its size as a continuous variable. Future studies would benefit from evaluating the size of MVO to better understand its potential impact on strain analysis and myocardial function. Furthermore, the study did not assess changes in left atrial and ventricular strain parameters over time.
Additionally, since early gadolinium enhancement sequences are not routinely acquired in our institution, the use of late MVO rather than early MVO may have led to an underestimation of the extent of MVO.

Finally, no T2* sequences were routinely performed in our institution limiting the assessment of concomitant intra-myocardial haemorrhage. Future longitudinal research is needed to explore the prospective relationship between this CMR parameter and patient outcomes, as well as to investigate quantitative changes in atrial and ventricular function over time

Conclusion

In STEMI patients after reperfusion during the acute/subacute phase, the presence of MVO is independently linked to impairment of atrial and ventricular strain parameters, beyond LV ejection fraction and LGE extent suggesting myocardial damage that involves the remote myocardium, as well as an increased risk of in-hospital complications. Further longitudinal studies are required to confirm our results and evaluate changes in atrial and ventricular function during follow-up.

Conflicts of Interest: Nothing to Disclose.

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Data availability: All data are available from the corresponding author on request.

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Figure legends.

Figure 1: An example of MVO in a patient with STEMI. Panel A displays a short-axis LGE image of an anterior STEMI with MVO (arrow in Panel A). Panel B presents the long-axis 2-chamber view LGE image of the same patient, with MVO highlighted by an arrow.
Abbreviations: LGE: Late gadolinium enhancement, MVO: Microvascular obstruction, STEMI: ST-segment elevation myocardial infarction.

Figure 2: Representative atrial and ventricular strain parameters impairment in STEMI with and without MVO detected by late gadolinium enhancement images. Short-axis and 3-chamber LGE images in patients with STEMI involving the left anterior artery with MVO are shown (Panels A and B). Atrial and ventricular strain parameters and their corresponding curves are reported in Panels C, D, E, and F. Short-axis and 2-chamber LGE images in patients with STEMI involving the left coronary artery without MVO are shown in Panels G and H. Atrial and ventricular strain parameters and their corresponding curves are reported in Panels I, J, K, and L.

Table legends

Table 1: Baseline characteristics of patients with and without MVO.

Abbreviations: CAD coronary artery disease; LAD left anterior descending coronary artery; LCx = left circumflex coronary artery; MVO microvascular obstruction; RCA = right coronary artery

Table 2: CMR findings of reperfused STEMI patients with and without MVO

Abbreviations: BSA, body surface area; CAD coronary artery disease; EDV, end-diastolic volume; ESV, end-systolic volume; GCS, global circumferential strain; GLS global longitudinal strain; GRS, global radial strain; LGE, late gadolinium enhancement; MVO microvascular obstruction; LV, left ventricle; SV, stroke volume; RV, right ventricle.

Table 3: Univariables determinants of MVO in reperfused STEMI patients.

Abbreviations: BSA, body surface area; CAD coronary artery disease; EDV, end-diastolic volume; ESV, end-systolic volume; GCS, global circumferential strain; GLS global longitudinal strain; GRS, global radial strain; LGE, late gadolinium enhancement; MVO microvascular obstruction; LV, left ventricle; SV, stroke volume; RV, right ventricle.

Table 4: Multivariable logistic regression analysis of atrial and ventricular strain parameters for discrimination between patients with and without MVO. Multivariable model was adjusted for age, sex, cardiovascular risk factors, multivessel disease, left ventricle ejection fraction, and LGE extent.

Abbreviations: GCS, global circumferential strain; GLS global longitudinal strain; GRS, global radial strain; MVO microvascular obstruction.