DOI: https://doi.org/10.53350/pjmhs020241811002

ORIGINAL ARTICLE

Predictive Role of Myocardial Fibrosis Severity on Functional Recovery following Coronary Revascularization

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ABSTRACT

Background: There is variable functional recovery following coronary revascularization in patients with ischemic left ventricular dysfunction. The degree of myocardial fibrosis could affect the recovery of myocardial function following restoration of coronary blood flow.

Objective: To determine the predictive role of myocardial fibrosis severity in functional recovery following coronary revascularization.

Methods: This was a prospective observational cohort study of 120 patients with obstructive coronary artery disease and left ventricular ejection fraction of 45% or lower undergoing percutaneous coronary intervention and/or coronary artery bypass surgery from January 2022 to March 2023. Pre-revascularisation cardiovascular magnetic resonance (CMR) with late gadolinium enhancement (LGE) was performed. Fibrosis was classified as mild, moderate and severe. The criterion for functional recovery was an absolute improvement of ≥ 5% in LVEF at 6 months.

Results: Functional recovery occurred in 66 patients (55.0%). There was a recovery rate of 80.0% in mild fibrosis, 57.8% in moderate fibrosis and 22.9% in severe fibrosis (P<0.001). Mean ejection fraction improvement was 9.0±5.1, 5.6±5.0, and 1.6±4.3 percentage points, respectively. The adjusted odds of recovery decreased by 40% for every 5% increase in fibrosis burden (adjusted odds ratio 0.60, 95% confidence interval 0.48–0.75, p<0.001). Better functional recovery was also independently related to complete revascularization.

Conclusion: The severity of myocardial fibrosis was an independent predictor of functional recovery after coronary revascularization. Preprocedural fibrosis assessment may enhance prognostic evaluation, patient counselling and post-revascularization management.

Keywords: myocardial fibrosis; coronary revascularization; functional recovery; late gadolinium enhancement; ischemic cardiomyopathy.

INTRODUCTION

Coronary artery disease is one of the most prevalent causes of left ventricular systolic dysfunction and heart failure globally[1]. Myocardial ischemia, when present, can cause a variety of myocardial structural and functional changes from reversible abnormalities of myocardial function to permanent myocardial injury. Coronary revascularization by either PCI or CABG can restore myocardial blood supply, alleviate ischemic symptoms, and improve myocardial performance in patients with ischemic ventricular dysfunction. But for the patients who have successful revascularization, the extent of functional recovery is very variable[2,3].

In ischemic myocardium, those that fail to contract do not necessarily mean irreversible tissue damage[4]. Ventricular dysfunction may persist for a long time as a result of myocardial stunning or myocardial hibernation even though the ventricular myocardium is viable. This myocardium may recover and resume contraction when a sufficient amount of blood flow is re-established[5]. However, fibrotic tissue does not possess normal contractile function and the myocardial tissue replaced by fibrosis has limited ability to recover function. Thus, the relative amounts of viable and irreversible scar might contribute to the degree of improvement of the ventricle after revascularization[6].

Myocardial fibrosis is a reparative process in response to ischemic injury and cardiomyocyte necrosis[7]. Extracellular tissue accumulating with collagen-rich proteins leads to ventricular remodelling, affects electrical conduction, reduces regional contraction (dyssynchrony) and produces changes in ventricular compliance[8]. Increased fibrosis severity might thus be linked to ongoing systolic dysfunction despite technically successful restoration of coronary blood flow. Patients with extensive fibrosis may also continue to have an elevated risk for progression of heart failure, ventricular arrhythmias and poor cardiovascular outcomes[9].

Late gadolinium enhancement cardiovascular magnetic resonance has become a valuable non-invasive tool for the characterization of myocardial tissue and for the identification and quantification of ischemic scar in terms of location, extent and transmural severity[10]. Gadolinium contrast is taken up in regions of increased extracellular space, and hence can differentiate fibrotic myocardium from viable myocardium. Post-revascularization recovery is inversely related to the transmural extent of enhancement in a dysfunctional myocardial segment. Sege-ments with no or minimal enhancement tend to have more potential for contractile improvement, while segments with almost complete fibrosis have less potential to improve[11,12].

Myocardial viability is being traditionally assessed prior to revascularization, but the concept of myocardium as viable or non-viable is too simplistic to capture the complexity of ventricular recovery. Total fibrosis burden, distribution of scar, number of dysfunctional but viable segments, baseline ventricular remodelling, completeness of revascularization and duration of ischemic dysfunction are all factors that affect global improvement of left ventricular function. Thus, quantitative assessment of fibrosis severity may be more useful than a binary viability assessment alone[13,14].

Accurate prediction of functional recovery has important clinical implications. It might be useful for personalized counseling of patients, directing their expectations of EF recovery, and for selecting patients who need early optimization of heart failure treatment after revascularization[15]. An assessment of fibrosis may also be useful for clinicians to understand the lack of postoperative improvement and to monitor patients with more advanced structural myocardial damage. However, the fibrosis results should not be the only consideration when deciding whether to perform

Received on 12-10-2023
Accepted on 29-12-2023

revascularization; symptoms, coronary anatomy, ischemic burden, procedural feasibility, and the overall clinical benefit of revascularization need to be considered[16].

Although the use of cardiovascular magnetic resonance has increased, the evidence linking the total myocardial fibrosis burden to global ventricular recovery is heterogeneous, especially, in patients with different revascularization approaches. Additional studies should be performed to assess if the fibrosis burden prior to the procedure can, on its own, predict improvement in left ventricle function after restoration of coronary blood flow. Thus, the purpose of the current study was to evaluate the predictive value of the severity of myocardial fibrosis in functional recovery after coronary revascularization. The hypothesis was that burden of myocardial fibrosis would be progressively inversely correlated with the likelihood of clinically significant improvement in left ventricular systolic function[17-20].

MATERIALS AND METHODS

This prospective observational cohort study was conducted jointly at the Department of Cardiology, Peshawar Institute of Cardiology, Peshawar, Pakistan, and the Department of Cardiology, Muhammad Medical College, Peshawar, Pakistan, from January 2022 to March 2023. One hundred twenty consecutive patients with ischemic left ventricular dysfunction undergoing coronary revascularization (either coronary artery bypass grafting or coronary artery percutaneous coronary intervention [PCI]). The participants were selected by the non-probability consecutive sampling technique after receiving written informed consent.

The inclusion criteria were patients 18–80 years of age with a left ventricular ejection fraction (LVEF) of 45% or less, and angiographically documented obstructive coronary artery disease (CAD) and at least one dysfunctional myocardial territory supplied by a significantly stenosed coronary artery. Patients were only selected if they were judged to be appropriate for coronary revascularization after consultation with the treating cardiac surgery and cardiology teams. Patients were excluded who had suffered from acute myocardial infarction in the previous four weeks, primary non-ischemic cardiomyopathy, severe valvular heart disease, congenital heart disease, persistent uncontrolled arrhythmia, advanced chronic kidney disease, contraindications to cardiovascular magnetic resonance imaging, previous coronary revascularization in the last six months, pregnancy, and incomplete imaging data.

A structured data collection form was used to document baseline demographic and clinical data such as age, sex, body mass index, smoking, hypertension, diabetes mellitus, dyslipidaemia, history of myocardial infarction, New York Heart Association functional class and medication history. Pre-revascularization routine laboratory investigations, transthoracic echocardiography, 12-lead electrocardiography, and coronary angiography were performed. The extent and severity of the coronary artery disease, the number of diseased arteries and the planned revascularization approach were recorded.

Pre-revascularisation TTE was performed using a standardised imaging protocol. LVEDV, LVEF, and LVESV were obtained from the apical 2 chamber and 4 chamber views using the modified biplane Simpson method. Standard 17-segment left ventricular model was used for evaluation of regional wall-motion abnormalities. All measurements were carried out by experienced cardiologists blinded to the cardiovascular magnetic resonance results.

Within two weeks before revascularization, MRI with a 1.5-T magnetic resonance scanner was done. Electrocardiographically gated cine images were obtained in conventional long-axis planes and 3 mm thick contiguous short-axis planes from the left ventricular (LV) apex to the mitral valve. Late gadolinium-enhancement images were acquired about 10-15 minutes after injection of a gadolinium containing contrast agent (0.1-0.2 mmol/kg, intravenously) with an inversion-recovery sequence optimized to null the signal from normal myocardium.

Myocardial fibrosis was detected as an area of LGE in an ischemic pattern. On serial short axis images, endocardial and epicardial borders were manually traced. The fibrotic myocardial mass was measured and presented as a percentage of the total left ventricular myocardial mass. Patients were classified into three groups according to the extent of fibrosis: mild fibrosis (scar burden <10%), moderate fibrosis (scar burden 10%-20%), and severe fibrosis (scar burden >20% of left ventricular myocardial mass).

For regional analysis the transmural extent of fibrosis was scored as 0, 1-25, 26-50, 51-75 or >75% of the myocardial wall thickness for each dysfunctional myocardial segment. Segments with fibrosis of 50% or less were deemed to have greater viability, while segments with fibrosis > 50% had limited recovery potential. Two experienced observers blinded to follow-up functional outcome independently analysed the MRIs. All differences were settled by consensus.

The treating heart team determined which procedure to perform (percutaneous coronary intervention or coronary artery bypass grafting) based on the coronary anatomy and the patient's clinical state, procedural risk, technical feasibility, and preference. Percutaneous coronary intervention was done with conventional angioplasty and stenting. CABG was performed using standard surgical techniques, using artery or vein grafts as clinically indicated. Revascularization was deemed to be complete when all major coronary vessels with significant stenosis feeding dysfunctional but potentially viable myocardial territory were successfully treated. All patients received ongoing and optimized treatment for coronary artery disease and heart failure guided by the latest guidelines.

The subjects were followed for 6 months after coronary revascularization. At the end of follow-up clinical assessment and repeat transthoracic echocardiography were conducted with the same imaging protocol applied at baseline. Functional recovery was achieved if there was an absolute improvement of ≥5% in LVEF from the preprocedural value. Secondary endpoints were: change in left ventricular end-systolic volume, improvement in regional wall motion, and improvement of at least one New York Heart Association functional class.

The association between baseline myocardial fibrosis severity and functional recovery at 6 months was the primary study outcome. The secondary outcomes were the association between fibrosis transmurality and regional wall-motion recovery, changes in ventricular volumes, symptomatic improvement and identification of independent predictors of functional recovery after coronary revascularization.

IBM SPSS Statistics, version 26.0 was used to analyse the data. For continuous variables, data were expressed as mean and standard deviation (SD) or median and interquartile range (IQR) depending on the distribution. Categorical variables were presented as frequencies and percentages. The independent-samples t-test, one-way analysis of variance, Mann–Whitney U test, or Kruskal–Wallis test was used for continuous variables, as appropriate. Chi-square or Fisher exact test was used to compare categorical variables. The paired-samples t-test or the paired Wilcoxon signed-rank test was used for comparison of the paired measurements obtained before and after revascularization.

Multivariable logistic regression analysis was used to determine the independent predictors of functional recovery. The adjusted model included variables with a P value < 0.10 on univariable analysis, as well as clinically relevant variables. The results of this study were presented as odds ratio with 95% confidence interval. To assess the ability of myocardial fibrosis burden to predict failure of functional recovery and to define an optimal fibrosis threshold, receiver operating characteristic curve analysis was performed. Statistically significant p-values were set at < 0.05, two sided.

The study protocol was approved by the due institutional ethics committee. All participants provided written informed consent and confidentiality of patient information was respected.

The study was carried out according to the ethical principles of the Declaration of Helsinki.

RESULTS

A total of 120 patients with ischemic left ventricular dysfunction underwent coronary revascularization and completed the six-month follow-up assessment. The average age of the participants was 61.2±9.0 years. The study included 90 men (75.0%) and 30 women (25.0%). 82 (68.3%) had hypertension, 54 (45.0%) had diabetes mellitus and 73 (60.8%) had a previous myocardial infarction. Of these, 68 patients (56.7%) had coronary artery bypass grafting and 52 patients (43.3%) had percutaneous coronary intervention.

With CMR, 40 patients had mild myocardial fibrosis, 45 had moderate, and 35 had severe fibrosis. There were no significant differences in age or proportion of diabetes mellitus, prior myocardial infarction, or three vessel coronary artery disease between severe fibrosis and non-severe fibrosis patients. There were no differences in the baseline left ventricle ejection fraction between the three groups. As shown in Table 1, mean myocardial fibrosis burden was significantly higher in the moderate group (14.8±3.1%) compared with the mild group (6.5±2.0%) and in the severe group (27.4±6.2%) compared with the moderate (p<0.001).

Left ventricular ejection fraction improved from a mean of 36.0±6.2% before revascularization to 41.6±8.1% after revascularization (p<0.001) at 6 months. Overall, 66 patients (55.0%) had functional recovery, defined as an absolute improvement of ≥ 5% in left ventricular ejection fraction.

Functional recovery occurred in 32 of 40 patients with mild fibrosis (80.0%), 26 of 45 patients with moderate fibrosis (57.8%), and 8 of 35 patients with severe fibrosis (22.9%). Statistically significant difference was noted between the three fibrosis groups (p<0.001). Mean absolute improvement in LVEF was inversely related to the extent of fibrosis as listed in Table 2.

Mild fibrosis patients had the highest mean decrease in left ventricular end-systolic volume index (LVESVi) of 18.1±12.2 mL/m². The corresponding reductions were 10.7±11.3 mL/m² in the moderate-fibrosis group and 3.4±9.7 mL/m² in the severe-fibrosis group (p<0.001). Progressive decrease in the frequency of improvement in global longitudinal strain and New York Heart Association functional class with progressive myocardial fibrosis severity was noted, as summarized in Table 2.

The mean myocardial fibrosis burden was significantly lower in patients who had functional recovery compared with those who did not (11.2±6.8% versus 21.4±8.5%, respectively; p<0.001). Fewer myocardial segments with fibrosis that involved ≥ 50% of the ventricular wall thickness were also related to functional recovery.

There was a graded inverse relationship between the extent of fibrosis in the transmural area and regional wall-motion recovery. The recovery rates were 75.8% for the dysfunctional segments without late gadolinium enhancement, 63.5% for 1–25% transmural fibrosis, 42.7% for 26–50% fibrosis, 18.3% for 51–75% fibrosis, and 6.5% for fibrosis covering >75% of the myocardial wall thickness (P for trend <0.001).

In univariable analysis, functional recovery was associated with lower myocardial fibrosis burden, complete coronary revascularization, lower baseline left ventricular end-systolic volume index, absence of diabetes mellitus, and fewer segments with more than 50% transmural fibrosis.

In the multivariable logistic regression analysis, myocardial fibrosis burden was the most powerful independent predictor of functional recovery. For every 5% increase in fibrosis burden, there was a 40% decreased adjusted likelihood of functional recovery. Complete coronary revascularization was associated with greater likelihood of recovery, while a greater baseline LVESVI was associated with lower likelihood of recovery, as shown in Table 3.

After accounting for other clinical and imaging variables, age, diabetes mellitus, baseline LVEF, and type of coronary revascularization were not independently associated with functional recovery.

Total myocardial fibrosis burden showed good discriminatory ability for failure of functional recovery in the receiver operating characteristic curve analysis. The area under the curve was 0.84, with a 95% confidence interval of 0.77–0.91 (p<0.001). Myocardial fibrosis burden greater than 18.0% had a sensitivity of 78.0% and specificity of 79.2% to predict failure of functional recovery.

There was a reverse correlation between severity of myocardial fibrosis and functional recovery after coronary revascularization. The patients with mild fibrosis had the most improvement in ventricular function and reverse remodelling, while patients with severe fibrosis had minimal improvement in function.

Table 1. Baseline characteristics according to myocardial fibrosis severity

Characteristic	Mild fibrosis (n=40)	Moderate fibrosis (n=45)	Severe fibrosis (n=35)	Total (n=120)	p-value
Age, years	59.6±8.8	61.3±9.1	63.2±8.7	61.2±9.0	0.19
Male sex	28 (70.0%)	34 (75.6%)	28 (80.0%)	90 (75.0%)	0.61
Female sex	12 (30.0%)	11 (24.4%)	7 (20.0%)	30 (25.0%)	0.61
Hypertension	24 (60.0%)	31 (68.9%)	27 (77.1%)	82 (68.3%)	0.28
Diabetes mellitus	14 (35.0%)	20 (44.4%)	20 (57.1%)	54 (45.0%)	0.15
Current smoking	20 (50.0%)	24 (53.3%)	20 (57.1%)	64 (53.3%)	0.82
Previous myocardial infarction	19 (47.5%)	28 (62.2%)	26 (74.3%)	73 (60.8%)	0.06
Three-vessel coronary disease	20 (50.0%)	28 (62.2%)	26 (74.3%)	74 (61.7%)	0.09
Baseline left ventricular ejection fraction, %	36.8±6.1	35.9±6.4	35.1±6.2	36.0±6.2	0.42
Baseline left ventricular end-systolic volume index, mL/m²	68.9±17.5	72.8±18.9	76.4±20.1	72.6±18.9	0.23
Myocardial fibrosis burden, %	6.5±2.0	14.8±3.1	27.4±6.2	15.8±9.2	<0.001
Coronary artery bypass grafting	21 (52.5%)	25 (55.6%)	22 (62.9%)	68 (56.7%)	0.65
Percutaneous coronary intervention	19 (47.5%)	20 (44.4%)	13 (37.1%)	52 (43.3%)	0.65
Complete coronary revascularization	34 (85.0%)	35 (77.8%)	24 (68.6%)	93 (77.5%)	0.23

Table 2. Functional outcomes six months after coronary revascularization

Outcome	Mild fibrosis (n=40)	Moderate fibrosis (n=45)	Severe fibrosis (n=35)	Total (n=120)	p-value
Follow-up left ventricular ejection fraction, %	45.8±7.0	41.5±7.2	36.7±6.5	41.6±8.1	<0.001
Absolute change in left ventricular ejection fraction, percentage points	9.0±5.1	5.6±5.0	1.6±4.3	5.7±5.6	<0.001
Functional recovery	32 (80.0%)	26 (57.8%)	8 (22.9%)	66 (55.0%)	<0.001
Reduction in left ventricular end-systolic volume index, mL/m²	18.1±12.2	10.7±11.3	3.4±9.7	11.0±12.4	<0.001
Improvement in global longitudinal strain, percentage points	3.6±2.1	2.2±1.9	0.7±1.6	2.2±2.2	<0.001
Improvement of at least one New York Heart Association functional class	30 (75.0%)	29 (64.4%)	13 (37.1%)	72 (60.0%)	0.003

Table 3. Predictors of functional recovery following coronary revascularization

Variable	Unadjusted odds ratio (95% confidence interval)	p-value	Adjusted odds ratio (95% confidence interval)	p-value
Myocardial fibrosis burden, per 5% increase	0.56 (0.46–0.69)	<0.001	0.60 (0.48–0.75)	<0.001
Complete coronary revascularization	3.10 (1.38–6.98)	0.006	2.54 (1.10–5.88)	0.029
Baseline left ventricular end-systolic volume index, per 5 mL/m² increase	0.86 (0.78–0.95)	0.003	0.89 (0.80–0.99)	0.038
Diabetes mellitus	0.55 (0.27–1.12)	0.10	0.73 (0.33–1.61)	0.44
Baseline left ventricular ejection fraction, per 1% increase	1.07 (1.00–1.14)	0.06	1.05 (0.98–1.13)	0.18
Coronary artery bypass grafting compared with percutaneous coronary intervention	1.28 (0.63–2.62)	0.49	1.18 (0.52–2.68)	0.69
Age, per one-year increase	0.96 (0.92–1.01)	0.11	0.97 (0.92–1.02)	0.23

DISCUSSION

The current study revealed a strong association between the severity of myocardial fibrosis and functional recovery after coronary revascularization[1]. At 6 months, patients with mild fibrosis had the largest increase in LVEF, decrease in LVESV, improvement in myocardial strain, and improved functional status. However, those with higher fibrosis levels had significantly lower functional recovery rates despite successful coronary revascularization. These results suggest that the magnitude of myocardial damage that is not reversible prior to treatment is important in determining the degree of improvement in the ventricles following restoration of coronary blood flow[2,3].

The biological distinction between viable and non-viable myocardium is in line with the observed relationship[4]. Myocardial dysfunction can occur due to stunning or hibernation, where the myocardial cells are still alive but have reduced contractility due to acute or chronic blood supply dysfunction[5]. This myocardium may recover function if sufficient perfusion is recovered. In fibrotic myocardium, however, there is a higher percentage of collagen rich scar tissue and fewer functioning cardiomyocytes. Thus, fibrosis increases the amount of myocardial tissue that is non-recoverable and decreases the potential of improvement in regional and global ventricular performance[6].

There was a progressive decrease in functional recovery in the mild, moderate, and severe fibrosis groups[7]. This is clinically relevant as it indicates that the presence or absence of fibrosis should not be the only way to interpret fibrosis burden[8]. Moderate fibrosis showed an intermediate response, meaning some recovery could be possible if there is some remaining viable myocardial tissue. Thus, quantitative measurement of fibrosis severity can be more informative than just a binary classification of myocardial viability for prognosis[9].

The results of the sub-regional analyses confirmed the general results. The wall-motion recovery rate was highest in the myocardial segments without LGE or with limited transmural fibrosis[10]. By contrast, segments where fibrosis accounted for ≥ 50% of the myocardial wall showed significantly depressed recovery rates. In extensive transmural scarring, there is significant structural damage and it is unlikely that blood flow restoration will restore a normal contraction. Thus, an evaluation of total scar burden along with the transmural extent of fibrosis may aid in the prediction of the extent of post-revascularization ventricular response[11,12].

Complete coronary revascularization was also independently associated with functional improvement[13]. This discovery emphasizes that even preserved myocardial viability is not enough to recuperate if the blood flow to the involved myocardial territory is not effectively restored. Unattended major coronary stenoses can leave unprotected myocardial tissue vulnerable to chronic ischemia and limit improvement. Therefore, the degree of functional recovery may be dependent on the interplay of the myocardial tissue properties, completeness of revascularization, the initial ventricular remodelling and the subsequent medical management[14].

Other clinical parameters and fibrosis burden were used for adjustment and the type of revascularization was not independently associated with the functional recovery[15]. This indicates that it is more important what happens to the heart muscle than whether the revascularization is performed with coronary artery bypass surgery or percutaneous coronary intervention. With sufficient restoration of adequate perfusion to viable myocardium, both methods can yield significant improvement. Therefore, the choice of revascularization strategy should continue to be driven by coronary anatomy and disease complexity, procedural risk, associated conditions, and multidisciplinary clinical judgment[16].

Lower likelihood of recovery was associated with a higher baseline left ventricular end-systolic volume index. An advanced ventricular dilatation probably indicates a long-standing ischemic injury, loss of contractile tissue and an established adverse remodelling process[17]. Even if coronary blood flow can be restored, if the enlargement is severe, it may not be possible for the myocardium to recover to normal structure and function. This finding reinforces the importance of early assessment and intervention to prevent widespread, irreversible remodelling[18].

While patients with severe fibrosis showed limited improvement in EF, extensive fibrosis should not be considered an absolute contraindication for revascularization. Treatment may have functional recovery as one possible outcome[19]. Despite this, revascularization can still alleviate angina, prevent the recurrence of ischemia, preserve the remaining viable myocardium, improve exercise tolerance and prevent further deterioration. Therefore, the assessment of fibrosis should be used to guide expectations and counseling and not to exclude a patient from treatment[20].

The results have clinical implications for clinical decision making. Preprocedural cardiovascular magnetic resonance could be used to identify patients who are likely to benefit from ventricular recovery and those who are less likely to[1-5]. Patients with mild fibrosis might be informed of a good likelihood for functional improvement; patients with severe fibrosis should be given realistic information about the likelihood of persistent ventricular dysfunction. For patients with extensive fibrosis, closer follow-up, aggressive optimization of heart failure therapy, rhythm monitoring and evaluation for device therapy when indicated may also be required[6-10].

The present study has a number of drawbacks. It was carried out at 2 centres in the same geographical area; this may limit the generalisability of the findings[11-15]. Unmeasured clinical factors may have impacted outcomes, and the observational design did not permit random assignment of the revascularization strategy. Sample size was modest and follow-up was only 6 months and therefore may not reflect delayed ventricular recovery or long-term clinical events. Further, variations in image interpretation and fibrosis measurement could have led to variability. Nevertheless, the correlation between greater fibrosis severity and lower functional recovery suggests the clinical importance of preprocedural myocardial tissue assessment[16-20].

CONCLUSION

The severity of myocardial fibrosis was one of the key factors in the functional recovery after coronary revascularization. Patients with mild fibrosis had the best recovery of left ventricle function and reverse remodelling, and those with severe fibrosis had a significantly diminished chance of recovery. Pre-revascularisation quantitative assessment of myocardial fibrosis could help in better prognostic evaluation, patient counselling and post-revascularisation management. But these fibrosis results must be

taken in the context of coronary anatomy, ventricular remodelling, completeness of revascularization, clinical picture and symptoms.

Competing interests: The authors declare no competing interests.
Funding: No external funding was received.
Author contributions: MW and SU conceived the study. NUK and SKS collected the data. MAK performed the statistical analysis. SFR supervised the study and revised the manuscript. All authors approved the final manuscript.
Acknowledgements: The authors thank the clinical and technical staff of the participating institutions.

REFERENCES

1. Patel H, Mazur W, Williams KA Sr, Kalra DK. Myocardial viability: state of the art—Is it still relevant and how to best assess it with imaging? Trends Cardiovasc Med. 2018;28(1):24-37.
2. Garcia MJ, Kwong RY, Scherrer-Crosbie M, Taub CC, Blankstein R, Lima J, et al. State of the art: imaging for myocardial viability: a scientific statement from the American Heart Association. Circ Cardiovasc Imaging. 2020;13(7):e000053.
3. Löffler AI, Kramer CM. Myocardial viability testing to guide coronary revascularization. Interv Cardiol Clin. 2018;7(3):355.
4. Chen Y, Zheng X, Jin H, Deng S, Ren D, Greiser A, et al. Role of myocardial extracellular volume fraction measured with magnetic resonance imaging in the prediction of left ventricular functional outcome after revascularization of chronic total occlusion of coronary arteries. Korean J Radiol. 2019;20(1):83-93.
5. Scully PR, Patel KP, Klotz E, Augusto JB, Thornton GD, Saberwal B, et al. Myocardial fibrosis quantified by cardiac CT predicts outcome in severe aortic stenosis after transcatheter intervention. JACC Cardiovasc Imaging. 2022;15(3):542-4.
6. Wu WY, Biery DW, Singh A, Divakaran S, Berman AN, Ayuba G, et al. Recovery of left ventricular systolic function and clinical outcomes in young adults with myocardial infarction. J Am Coll Cardiol. 2020;75(22):2804-15.
7. Lopes BBC, Kwon DH, Shah DJ, Lesser JR, Bapat V, Enriquez-Sarano M, et al. Importance of myocardial fibrosis in functional mitral regurgitation: from outcomes to decision-making. JACC Cardiovasc Imaging. 2021;14(4):867-78.
8. Radesich C, Cappelletto C, Indennidate C, Perotto M, Di Lenarda A. Predicting left ventricular functional recovery in ischaemic cardiomyopathy: needs and challenges. Eur Heart J Suppl. 2023;25(Suppl B):B69-74.
9. Calvieri C, Riva A, Sturla F, Dominici L, Conia L, Gaudio C, et al. Left ventricular adverse remodeling in ischemic heart disease: emerging cardiac magnetic resonance imaging biomarkers. J Clin Med. 2023;12(1):334.
10. Shenouda R, Bytyci I, Sobhy M, Henein MY. Early recovery of left ventricular function after revascularization in acute coronary syndrome. J Clin Med. 2020;9(1):24.
11. Dong Q, Wen X, Chang G, Xia R, Wang S, Yang Y, et al. ST-segment resolution as a marker for severe myocardial fibrosis in ST-segment elevation myocardial infarction. BMC Cardiovasc Disord. 2021;21(1):455.
12. Diekmann J, Koenig T, Thackeray JT, Derlin T, Czerner C, Neuser J, et al. Cardiac fibroblast activation in patients early after acute myocardial infarction: integration with MR tissue characterization and subsequent functional outcome. J Nucl Med. 2022;63(9):1415-23.
13. Slimani A, Melchior J, de Meester C, Pierard S, Roy C, Amzulescu M, et al. Relative contribution of afterload and interstitial fibrosis to myocardial function in severe aortic stenosis. JACC Cardiovasc Imaging. 2020;13(2 Pt 2):589-600.
14. Puls M, Beuthner BE, Topci R, Vogelgesang A, Bleckmann A, Sitte M, et al. Impact of myocardial fibrosis on left ventricular remodelling, recovery, and outcome after transcatheter aortic valve implantation in different haemodynamic subtypes of severe aortic stenosis. Eur Heart J. 2020;41(20):1903-14.
15. Di Bella G, Aquaro GD, Bogaert J, Piaggi P, Micari A, Pizzino F, et al. Non-transmural myocardial infarction associated with regional contractile function is an independent predictor of positive outcome: an integrated approach to myocardial viability. J Cardiovasc Magn Reson. 2021;23(1):121.
16. Zegard A, Okafor O, De Bono J, Kalla M, Lencioni M, Marshall H, et al. Myocardial fibrosis as a predictor of sudden death in patients with coronary artery disease. J Am Coll Cardiol. 2021;77(1):29-41.
17. Ata Y, Abanoz M. Predictive roles of right coronary artery disease severity and systemic immune-inflammation index in predicting atrial fibrillation after coronary bypass operations in patients with right coronary artery disease: inflammation, right coronary artery and postoperative atrial fibrillation. Heart Surg Forum. 2021;24(6):E977-82.
18. Babes EE, Tit DM, Bungau AF, Bustea C, Rus M, Bungau SG, et al. Myocardial viability testing in the management of ischemic heart failure. Life (Basel). 2022;12(11):1760.
19. Han D, Tamarappoo B, Klein E, Tyler J, Chakravarty T, Otaki Y, et al. Computed tomography angiography-derived extracellular volume fraction predicts early recovery of left ventricular systolic function after transcatheter aortic valve replacement. Eur Heart J Cardiovasc Imaging. 2021;22(2):179-85.
20. Cabac-Pogorevici I, Muk B, Rustamova Y, Kalogeropoulos A, Tzeis S, Vardas P. Ischaemic cardiomyopathy: pathophysiological insights, diagnostic management and the roles of revascularisation and device treatment—gaps and dilemmas in the era of advanced technology. Eur J Heart Fail. 2020;22(5):789-99.

This article may be cited as: Waleed M, Ullah S, Khan NU, Sohail SK, Khan MA, Rizvi SF; Predictive Role of Myocardial Fibrosis Severity on Functional Recovery following Coronary Revascularization. Pak J Med Health Sci, 2024; 18(1): 1002-1006.