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Review Article

Cardiometabolic index as a predictor of left ventricular remodeling post myocardial infarction

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ABSTRACT

Myocardial Infarction has left an imprint on the heart in the form of left ventricular remodeling, which is the most important factor in the progression of heart failure and the gaining or losing of long-term cardiovascular prognosis. Traditional risk assessment has a narrow vision on metabolic factors which are most influential in the process of cardiac repair after the event. The cardiometabolic index, a composite of central adiposity along with dyslipidemia, is being recognized as a very easy and simple to use marker that reflects the metabolic stress situation. Observational studies have reported that increased cardiometabolic index is associated with unfavorable left ventricular structural changes such as concentric remodeling and hypertrophy along with decreased diastolic function indicating its use in predicting post-myocardial infarction outcomes. However, the majority of the data available so far come from studies of the general population rather than myocardial infarction-specific groups; yet the reasoning behind the mechanistic pathway is strong enough to support its presence in the post-infarction context where it provides a biologically plausible link between metabolic dysregulation and unfavorable changes in ventricle remodeling. This review presents the current evidence regarding cardiometabolic index and left ventricular remodeling, discusses its role in risk stratification and recognizes the lack of research in this regard. In order to prove cardiometabolic index as a predictor to assess its application in directing personalized interventions aimed at enhancing myocardial recovery, and to increase the associated life span of patients with heart problems, future clinical trials focusing on post-myocardial infarction patients are absolutely necessary.

Keywords: Cardiometabolic index, Insulin resistance, Left ventricular remodeling, Myocardial infarction, Post-infarction outcomes

INTRODUCTION

Myocardial infarction (MI), despite the fact that in some places it is treated and managed very well, still ranks among the top non-communicable diseases worldwide and continues to be one of the main causes of cardiovascular death and suffering.[1] Short-term outcomes have improved but still a large number of MI survivors develop left ventricular (LV) remodeling which is an irreversible process characterized by the gradual change in size, shape, and function of the ventricle.[2] LV remodeling, though a structural consequence, is not a benign one, and it has great clinical importance. It is moreover quite obviously linked to heart failure, significant arrhythmias, and a decrease in overall survival.[3] It has been reported that the left ventricle remodeling after a myocardial infarction consists of a number of intertwined biological processes concerning the death of myocardial tissue through inflammation, neurohormonal activities, extracellular matrix remodeling and myocardial reconfiguration.[4] Although the infarcted tissue size, the timing of reperfusion and the resultant hemodynamic stress are known factors, these often are insufficient to account for the remodeling divergence in

patients with the same myocardial infarction characteristics.[5] The remodeling divergence in myocardial infarction suggests that more systemic factors beyond the necrotic myocardial tissue are important in determining myocardial remodeling and its response post infarction.[6] The remodeling divergence has been coupled with the presence of cardiometabolic dysfunction, which has been identified as a significant factor in the ischemic heart disease continuum and adverse cardiovascular events.[7]

The metabolic abnormalities such as insulin resistance, excessive visceral fat and atherogenic dyslipidemia push the body toward a state of chronic inflammation, oxidative stress and so on, which eventually results in impaired myocardial energy handling.[8] These changes may not only adversely affect ischemic damage but also impair the phenomenon of adaptive remodeling during post-MI healing.[9] Conventional anthropological techniques and body mass index do not take into consideration visceral adiposity and its associated metabolic risk factors.[10] Therefore, people who have normal body weight but negative metabolic profiles may go unrecognized by customary risk evaluation methods.[11] This has resulted in increased interest in using combined indices, which provide a clearer indication of metabolic factors and dysfunction in adipose tissue than conventional assessing methods.[12]

The cardiometabolic index (CMI), which takes into consideration the waist-to-height ratio and the triglyceride-to-high-density lipoprotein cholesterol ratio to arrive at its score, is viewed as an exceptionally informative and at the same time user friendly criterion for visceral fat and metabolic disorder detection.[13] It has been discovered through substantial cohort investigations that high CMI is closely associated with the incidence of metabolic syndrome, ischemic heart disease and cardiovascular death.[14] There is a specific and significant relationship between CMI and these diseases, and that is the reason it has been dubbed the sensitive measure of the standard obesity related measures in different populations.[15] Moreover, the association of CMI with atherosclerotic disease risk is now being questioned as new studies reveal the close relationship between CMI and harmful cardiac structural remodeling.[16] Even after controlling for cardiovascular risk factors, patients with elevated levels of CMI exhibited unfavorable LV geometric patterns, like concentric remodeling and hypertrophy, which were significant.[17]

This finding supports the hypothesis that there is a direct relationship between metabolic stress on the whole body and improper cardiomyocyte structural changes.[18] In the case of patients who have had an MI, cardiometabolic abnormalities are very common and usually occur together with hypertension, diabetes and dyslipidemia.[19] However, modern post-MI risk assessment techniques still largely rely on clinical parameters, cardiac imaging results and circulating biomarkers, with little to no inclusion of composite metabolic indices.[20] Residual cardiometabolic risk, that is unfavorable LV remodeling and development of heart failure, might still be there and not accounted for through this method.[21] Consequently, if the CMI is included in post-MI evaluation, then it would be a chance for early detection of patients who are at the highest risk of learning the wrong way or experiencing bad healthcare outcomes, thus possibly improving the clinical outcome.[22]

MECHANISMS LINKING THE CARDIO-METABOLIC INDEX TO LEFT VENTRICULAR REMODELING AFTER MYOCARDIAL INFARCTION

Metabolic and inflammatory modulation of post-infarction myocardial repair

LV remodeling after a heart attack is a dynamic process of heart muscle repair that is very sensitive to systemic metabolic and inflammatory conditions.[23] It is known that the size of the scar tissue and the blood flow during reperfusion are the main factors influencing the early remodeling process; however, there is an increasing amount of evidence that points to the fact that cardiometabolic dysfunction plays a substantial role in the post-MI phases of healing and of the formation of ventricle dilation and fibrosis which, in turn determines whether the healing is adaptive or maladaptive.[24]

The CMI, which evaluates central obesity and atherogenic dyslipidemia together, indicates metabolic disturbances that may not end after the acute ischemic insult and that may influence the long-term ventricular remodeling paths.[25] Visceral fat is one of the main factors analyzed for CMI and assists the chronic low-grade inflammation model due to the secretion of excessive pro-inflammatory cytokines that could enhance the infarction inflammatory cascade and impair the process of myocardial injury repair.[26] The prolonged inflammatory activation after MI has been associated with the deposition of an extracellular matrix that is more than usual, the stiffening of the myocardium, and the gradual widening of the ventricle.[27] Recent studies have shown that a high CMI is linked to post-PCI coronary microvascular dysfunction in patients with acute STEMI, which points to the possibility of a connection among cardiometabolic burden, inflammation pathways and impaired myocardial perfusion. These processes might be regarded as the upstream factors that contribute to the bad changes in the left ventricle after a heart attack period.[28]

Insulin resistance, which is very much associated with visceral fat and high lipid levels, continues to interfere with post-MI myocardial healing process by altering the energy metabolism of the heart.[29] After the death of the heart muscle, the heart itself has to rely on flexible metabolism for tissue repair and functional recovery; however, the insulin resistance is consuming the substrate for energy in such a way that the less efficient and more harmful oxidation of the fatty acid is taking place, thus increasing the oxidative stress and impairing the function

of mitochondria.[30] The disturbance of these metabolic processes results in the death of the cardiomyocytes and the replacement by fibrosis not only in the infarcted area but also in the surrounding myocardial regions which are contributing to the unfavorable geometry of the left ventricle over time.[31]

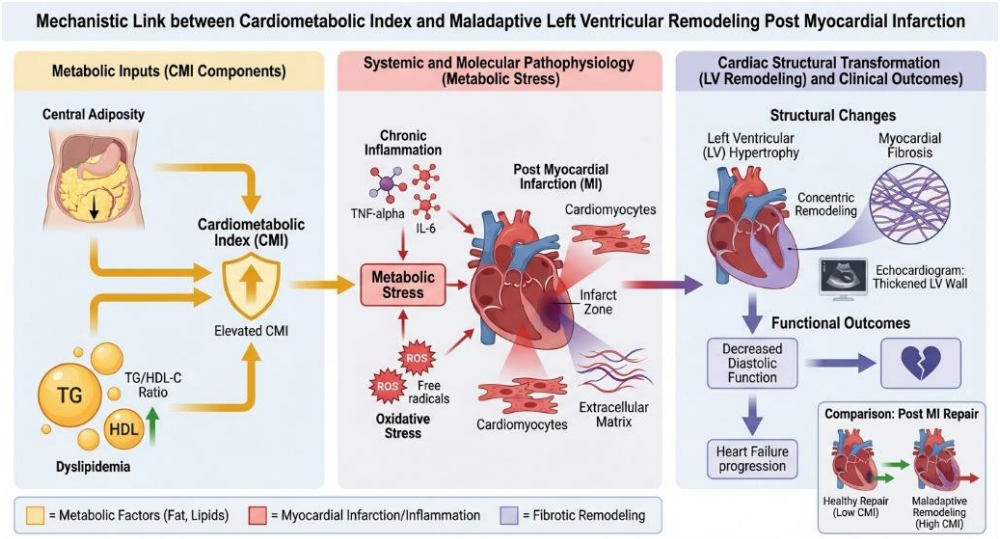

Figure 1: Mechanistic link between cardiometabolic index and maladaptive left ventricular remodeling post myocardial infarction.

Hemodynamic, neurohormonal and vascular mechanisms in post-MI remodeling

In addition to the heart's direct effects, the increased CMI which indicates cardiometabolic disorders also affect the remodeling of the heart after MI through the alteration of blood flow and the recruitment of hormones.[32] The combination showing the presence of atherogenic dyslipidemia and insulin resistance is marked by the loss of the endothelial barrier. It also involves a rise in the stiffness of the arteries. These changes leads to an increased afterload. As a result, the healing ventricle suffers additional mechanical stress.[33] The post-MI condition, where the vicious circle of the hemodynamic and metabolic stress being accompanied, accelerates the adverse ventricular remodeling and next drives pathological chamber dilatation and hypertrophy.[34]

The continuous stimulation of the renin angiotensin aldosterone system (RAAS) and sympathetic nervous system (SNS) after the death of heart tissue from lack of blood (infarction) is a main underlying mechanism that continues to work against the ventricular remodeling.[35] The continuous activation of the neurohormonal circuit fosters the heart muscle to become larger (hypertrophy), to develop more connective tissue (fibrosis), and to lose its pumping capacity (progressive ventricular dysfunction), and all these processes occur even when the patient is receiving medically directed therapy.[36] The neurohormonal activation in the post-myocardial infarction period, as characterized by blood-circulating biomarkers, is one of the major players in the deleterious ventricle remodeling and in its further development.[37] Epicardial fat tissue is another factor that acts as a physiological bridge between the burden of cardiometabolic and post-MI remodeling.[38] Excessively present epicardial fat tissue has been associated with the heart attack size increase and also the left ventricle systolic function decline in the post-myocardial infarction.[39] Besides, coronary microvascular dysfunction is a well-established consequence of cardiometabolic dysregulation that results in the lack of blood supply (perfusion) to the area around the infarct and to the non-infarcted myocardium. This condition results in mild ischemia, which further exaggerates the process of adverse remodeling.[40] Therefore, cardiometabolic dysfunction plays a central role as a potentiating factor that exacerbates adverse ventricular remodeling (dilation and hypertrophy) induced as a result of hemodynamic stresses following the infarction event.[41] This complex of pathways therefore clearly illustrates how the visceral fat and the associated atherogenic dyslipidemia induced by the CMI create a pathological environment that accelerates the key event drivers; the inflammation, metabolism, hemodynamic stresses and the neurohormonal activation associated with adverse post-infarction ventricular remodeling.

OVERVIEW OF CARDIOMETABOLIC INDICES IN THE POST–MYOCARDIAL INFARCTION SETTING

Cardiometabolic indices are gaining more importance in cardiology as composite indices of the combined burden of insulin resistance, central obesity, dyslipidaemia and systemic inflammation persisting after MI.[42] The traditional assessment of cardiac risk post-MI is primarily based on the infarct itself, the function of the left ventricle, as well as traditional factors of risk. However, analysis of

contemporary practice clearly shows that a sufficiently comprehensive integrated assessment of risk is not currently practiced in the clinic.[43] Body mass index (BMI) is the least sensitive anthropometric index for visceral fat assessment. It does not identify metabolically unhealthy patterns, which have a strong association with high cardiovascular risk.[44] Such a problem is of considerable importance for post-MI patients, in whom the process of myocardial repair and ventricular adaptation occurs against a background of a systemic metabolic environment, which can also affect the processes of inflammation, fibrosis or ventricular remodeling independent of the conventional factors.[45]

Various composite cardiometabolic indices have been developed that capture metabolic risk better than traditional methods using simple anthropometric and biochemical parameters.[46] TyG index showed to be a valid marker for insulin resistance in several studies. It is also related to worse cardiovascular outcome and impaired reperfusion after acute coronary syndrome.[47] The visceral adiposity index was developed to calculate visceral fat dysfunction and it was related to metabolic syndrome and subclinical cardiovascular disease.[48] In the same way, the product of lipid accumulation showed the excess of lipid accumulation in a particular region of the body and its association with hypertension in various populations. Systematic reviews revealed evidence of association between high LAP levels and hypertension.[49] Clearly, these indices reflect only one aspect of cardiometabolic risk. They highlight selected metabolic pathways and do not address the interaction between adiposity and dyslipidemia that is so critical to post-MI metabolic stress.[50] Thus, CMI provides an unusually relevant framework by combining the waist-to-height ratio as an indicator of central fat with the triglyceride-to-high-density lipoprotein cholesterol ratio as an indicator of the presence of insulin-resistant and atherogenic dyslipidemia.[51]

This combination makes CMI a mirror of the processes going on due to fat in the abdomen inflammation, lipid toxicity and dysfunction of the blood vessels and all these processes are responsible for the injury and remodeling in heart after the infarction.[52] High CMI has been shown to be independently related to the left ventricular structural patterns, which include concentric remodeling and hypertrophy, even though analysis including traditional cardiovascular risk factors has been performed.[53] Newer studies suggest that higher CMI may be linked to heart failure risk and increased mortality.[54] One of the possible mechanisms of this association could be vascular pathways because coronary microvascular dysfunction is a major factor leading to adverse outcomes in the case of HFpEF.[55] In the context of MI, the role of the residual metabolic stress in ventricular repair is crucial, in addition to the infarct size. Therefore, CMI is a biological measure with relevance in clinical practice as a risk stratification tool, in addition to giving information regarding vulnerability to left ventricular remodeling. Large CMI is related to poor microvascular outcomes of STEMI, as well as a significant mechanism for poor post-MI ventricular remodeling, such as microvascular dysfunction.[56] Apart from that, CMI has been found to have an independent link to increased heart failure risk after adjusting for traditional cardiovascular risk factors, which means it measures metabolic disturbances directly related to cardiopathology.[57] Research tells us of the correlation between higher CMI and adverse left ventricular geometric patterns including concentric remodeling and hypertrophy in population based studies.[58]

CLINICAL EVIDENCE LINKING THE CARDIOMETABOLIC INDEX TO LEFT VENTRICULAR REMODELING AND OUTCOMES

Through the use of observational studies involving both common and vulnerable cardiovascular populations, it has been confirmed that the association of higher CMI values with adverse LV structural remodeling was very strong after conventional cardiovascular risk factors such as age, hypertension, diabetes mellitus and body mass index were accounted.[59] Cardiometabolic burden being high, as indicated by composite markers corresponding with cardiometabolic index components, has been linked to an increase in left ventricular mass index which implies that the dysregulation of metabolism contributes to myocardial hypertrophic responses in addition to pressure overload alone.[60] CMI reflects that the highest level of metabolic burden was observed not only in the process of developing heart failure, but also in the risk factors that have been unveiled using the traditional measures of anthropometric values. Cardiometabolic burden, described as the measures of the metabolic profile reflective of CMI components, was found to be related to the thicker relative wall and the presence of concentric left ventricular remodeling and hypertrophy that represents higher cardiovascular risk.[61] On the other hand, these results make the case that CMI has more metabolic-related impacts on cardiovascular risk which are not sufficiently indicated by conventional measures like body mass index or waist circumference.[62]

Not only structural alterations but also high CMI levels are related to heart dysfunction that is not visible through clinical examination and to the greater chance of heart failure in people with normal systolic function.[63] This unrecognized diastolic dysfunction is the main sign of the heart's poor adaptation and its associated with the development of heart failure with preserved ejection fraction.[64] Despite a dearth of studies specifically evaluating CMI in post MI cohorts, compelling evidence from other cohorts with established coronary artery disease provides indirect support for its prognostic relevance.[65] A higher CMI has already been shown to be independently associated with hospitalization for heart failure in patients with Ischemic heart disease. This an important outcome clearly associated with adverse LV remodelling post MI.[66] The elevated levels of CMI has been linked to increased cardiovascular death in high-risk populations.[67] Further evidence shows that increased CMI

is related to coronary microvascular dysfunction. This is a pathological process that impairs myocardial perfusion. There is also a negative influence on infarct healing and ventricular remodelling.[68] Most importantly, the CMI measures the residual risk of CVD after considering the BMI, and CMI was found to have an independent association with CVD risk in this study. This predictive accuracy of CMI makes it highly valuable.[69] Collectively, the results simply that the CMI mirrors the higher metabolic stressors that are linked to the greater risk of negative cardiovascular events, thus indicating its possible application to reveal high-risk endotypes apart from the conventional metrics. Besides, these findings throw light on the necessity of future research that is to assess the role of CMI in post-infarction remodeling and risk classification through the use of prospective studies.[70]

CLINICAL IMPLICATIONS AND RISK STRATIFICATION IN POST-MYOCARDIAL INFARCTION PATIENTS

After MI, the risk stratification is still the most important factor in secondary prevention, which helps to find patients with the highest risk for negative LV remodeling, thus heart failure. Modern-day methods for evaluating patients at highest risk for an adverse outcome focus on only three important factors: infarct size, left ventricular ejection fraction, and neurohormonal markers. Nevertheless, the parameters mentioned above do not fully consider the extent to which systemic metabolic stress has a positive influence on myocardial repair and the long-term adaptation of the ventricle.[71] The combination of central adiposity and dyslipidemia synthesize CMI, which is an emerging marker. CMI is a clinically accessible marker combining central adiposity and dyslipidemia, two interconnected metabolic disorders that are more and more accepted as modifiers of cardiovascular risk with possibly effect on post-infarction remodeling.[72]

The CMI is a key clinical advantage because it can spot the remaining cardiometabolic risk among the patients who might still be categorized as low or medium risk with the use of the standard metrics. The CMI level has been correlated with the odds of having heart failure and coronary artery calcium, along with the risk of being newly diagnosed with cardiovascular disease and dying from cardiovascular causes being all the time high; thus, all of these signify the unified impact of central fat and dyslipidemia on disease pathways and confirm its role as an adjunctive marker in thorough risk assessment.[73] As the metabolic effects that the traditional anthropometric measures like BMI do not show perfectly well are captured by the elevated CMI, it may reveal the pronounced likelihood of the onset of the adverse ventricular remodeling that is caused by the whole body metabolic dysfunction.[74]

On top of that, CMI can help in the process of spotting the patients who are more likely to suffer from heart failure. This is because higher CMI levels have been shown to be a powerful and independent factor for heart failure risk in several population studies. Most of the time, such a situation has been regarded as a combined effect of visceral fat and the lipid profiles of atherogenic nature. CMI has been associated with these metabolic abnormalities, which result in heart failure, and thus, CMI can be considered as capturing upstream cardiometabolic determinants that are relevant to ventricular dysfunction in ischemic and post-MI populations.[75] The inclusion of CMI into post-infarction risk models might even lead to better and earlier recognition of the patients who could benefit from intensive monitoring or preventive measures.

From the practicality aspect, CMI has a number of characteristics that would render it fitting for potential regular clinical application. This metric incorporates usual height and weight measurements, along with lipid testing and includes central fatness and lipid disorder and is therefore a better assessment of overall CVD impact than individual measures such as waist circumferences and/or triglyceride levels.[76] It is these characteristics, that make CMI so attractive in following up patients who have been treated and released for outpatient care for an MI diagnosis, especially in low resource hospitals.

CMI may point the way to post-myocardial infarction risk stratification since a higher value of CMI indicates an unfavorable cardiometabolic profile that is highly connected with ischemic heart disease and previous myocardial infarction. The connection between cardiometabolic dysfunction and adverse ventricular remodeling is well established, thus, patients with high CMI might need closer echocardiographic monitoring after MI.[77] In this case, CMI may work hand-in-hand with imaging-based markers by helping to differentiate patients whose structural and functional alteration is more likely to happen over time.

Ultimately, the clinical significance of CMI reaches beyond the frontiers of the conventional model of personalized care in therapeutics related to cardiovascular medicine. Metabolic malfunction is being recognized as the key factor triggering the development of concentric remodeling patterns and heart failure phenotypes after MI, thus CMI can help to make further distinctions in the risk stratification models that are based on infarct-centric approaches.[78] Therefore, incorporating CMI into the post-infarction evaluation would make it possible to offer a well-rounded and personalized secondary prevention that would take into account both the ischemic injury and the systemic metabolic vulnerability.

LIMITATIONS AND CHALLENGES

The CMI as a marker of adverse cardiac remodeling and cardiovascular risk is getting more and more evidence-based support. However, there are still several very significant limitations. It is particularly noteworthy that the bulk of the CMI studies have been conducted in general or community-based populations, leaving those who have

gone through a MI out of the picture. This eventually leads to the situation where the generalization of the findings to the post-MI patients is highly questionable. Patients receiving treatment after MI constitute a special group with a high risk; thus, the differences in processes occurring in the body during treatment alone preclude making predictions regarding the prognostic significance of CMI in post-infarct left ventricular remodeling and heart failure. However, most modern research still relies on cross-sectional studies or observational studies, and the results are rather hard to interpret from a causal point of view. Of course, similarities in basic population characteristics, such as age, sex, ethnicity, and baseline cardiovascular risk factors, make generalization of results even more complicated. The inconsistency between studies may be also due to the different approaches to assessing CMI in terms of waist circumference and lipid measurement. In view of all these limitations, there is a dire need for well-framed prospective studies focusing exclusively on post-MI patients. In fact, the outcomes of such studies would help in clarifying not only the role of CMI in risk stratification but also the time and need for its early intervention in the population.

CONCLUSION

The CMI has come to the forefront as a strong yet uncomplicated marker that represents the interrelationship between central fatness and lipid defects two metabolic factors that greatly impact heart health. There is a growing body of evidence that connects high CMI with poor left ventricular remodeling and increased heart failure, thus implying its value in the identification of patients with myocardial infarction at risk of death.Nonetheless, the majority of the research done till now has centered on the general population, which has resulted in our poor understanding of CMI's predictive power in specifically post-MI patients. It becomes imperative to conduct future prospective studies to authenticate its importance in this particular situation and to clarify if metabolic health-improving interventions can have a positive impact on cardiac remodeling and clinical outcome. Incorporating CMI into the standard care of post-MI patients could provide cardiologists with a practical tool that allows them to better categorize risk, customize treatment and eventually extend a patient's cardiovascular prognosis in the long run. CMI promises to improve patient management and steer precision-directed strategies right after an infarction by linking metabolic factors with the assessment of cardiac structure.

RESEARCH OPPORTUNITIES AND FUTURE DIRECTIONS

Despite growing evidence on the association between the CMI and negative cardiovascular outcomes, there are still vast areas to be researched, particularly regarding MI survivors. Up to now, the majority of studies were conducted either on very low-risk or very high-risk populations, thus leaving the issue as to whether CMI can predict LV remodeling and heart failure after MI still unresolved. Hence, CMI should be tested in a specially designed prospective study in post-MI patients to ascertain if it can be used as an early risk stratification, prognosis, and therapy guidance tool.

The CMI measurement should be combined with advanced cardiac imaging techniques such as echocardiography and MRI to monitor the heart's structural and functional changes during the treatment period. Mechanistic studies that investigate the connection between CMI-derived metabolic disorders and central obesity, dyslipidemia and insulin resistance, which lead to myocardial fibrosis, inflammation, and mal-adaptive remodeling, could open up new targets for therapy.

Moreover, there is an exciting avenue of CMI research that involves investigating whether implementing lifestyle changes and drugs according to CMI can cut back on adverse remodeling and ultimately improve the quality of life for post-MI patients. Also, the combination of CMI with other new biomarkers may further improve the models of risk prediction, thus contributing to a more personalized and precision-based approach to cardiovascular care. Finally, the resolution of these issues will not only increase our awareness of CMI but also facilitate the application of this straightforward and clinically friendly metric in practice or on a large scale as a part of the strategies to enhance recovery and long-term outcomes after MI.

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