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Assessment of global longitudinal strain and plasma natriuretic peptide in patients with asymptomatic left ventricular dysfunction

Objective — The purpose of this study was to investigate the association between global longitudinal strain (GLS) and plasma NT-proBNP for predicting left ventricular (LV) performance in asymptomatic patients after acute myocardial infarction (AMI).

Material and methods — We prospectively included patients with diagnosis of AMI without clinical signs and symptoms of heart failure (HF) and followed these patients for 6 mos. Baseline echocardiography was performed at admission, and follow-up echocardiography was performed after 6 mos. A normal GLS was defined as having an absolute value of ≥16%. According to the baseline GLS, participants were divided into two groups and compared. In all participants, blood samples of plasma NT-proBNP were obtained at admission, before discharge, and 6 mo after discharge.

Results — The study population was consisted of 98 participants, of which 80 (81.6%) were males, and the mean age was 56.0±9.3 years. Baseline echocardiography showed that most of the participants (60, 61.2%) had abnormal GLS<16%, whereas 38 (38.8%) participants had normal or borderline GLS ≥16%. Compared with the normal GLS group, participants with abnormal GLS had higher GRACE score, higher troponin I concentration, lower systolic blood pressure, lower mean LV ejection fraction, and decreased LV diastolic function. At 6-mo follow-up, only LV systolic function remained significantly different between the two groups. Compared to baseline, there was a significant improvement of GLS in the abnormal GLS group at 6-mo follow-up (p=0.04). Prevalence of complications after AMI was significantly higher in this group. There were significant differences between baseline and discharge NT-proBNP concentrations between the two groups (p<0.05). In the abnormal GLS group, there were significant correlations between baseline and discharge NT-proBNP concentrations with baseline LV systolic function. Discharge NT-proBNP concentration also correlated significantly with 6-mo follow-up GLS. For determining the effect of baseline GLS abnormality, the areas under the ROC curve for baseline and discharge NT-proBNP concentrations were 0.73 (95% CI 0.60–0.85, p=0.001) and 0.77 (95% CI 0.66–0.87, p<0.001), respectively. Regarding early prediction of follow-up GLS abnormality, the area under the ROC curve for discharge NT-proBNP concentration was significantly higher 0.70 (95% CI 0.55–0.84, p=0.016). The optimum cut-off value of discharge NT-pro-BNP was 688.5 pg/ml, with 72.4% sensitivity and 65.4% specificity to predict 6-mon GLS abnormality following acute myocardial infarction.

Conclusion — The main finding of this study is that impaired LV GLS is associated with elevated plasma concentrations of NT-proBNP in post-AMI patients. Pre-discharge NT-proBNP concentration combined with impaired initial GLS could predict worsening LV systolic function over time in asymptomatic post-AMI patients.

Keywords — Myocardial infarction; natriuretic peptide; echocardiography; left ventricular dysfunction

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Introduction

In Mongolia, the incidence of cardiovascular disease, which is the first leading cause of mortality and the third leading cause of morbidity, has doubled over the last 20 yrs [1]. Among cardiovascular diseases, ischemic heart disease is the most prevalent [2]. Left ventricular (LV) systolic dysfunction is a frequent and severe complication of ischemic heart disease. It is particularly associated with myocardial infarction (MI), which increases the risk of sudden death and heart failure (HF) [3]. Asymptomatic LV systolic dysfunction (ALVSD)

is defined as depressed LV systolic function in the absence of signs and symptoms of clinical heart failure [4]. SAVE and TRACE trial results show that 40–58% of post-MI patients developed ALVSD. Post-infarction ALVSD is also associated with a higher risk of adverse events, including progressive HF and mortality [5, 6]. Thus, early recognition of ALVSD in such patients could reduce adverse clinical outcome.

Currently, in patients with acute MI (AMI), routine echocardiography is advised, and LV ejection fraction (LVEF) is a key parameter for assessing LV systolic function. In fact, recent studies have shown that imaging of global longitudinal strain (GLS) is more sensitive to ischemia and wall tension, and is able to detect irregular contraction patterns even with normal LVEF [7, 8]. Longitudinally directed, subendocardial fibers of LV myocardium are more susceptible to ischemia. Thus, subendocardial dysfunction in early ischemia would be expected to affect longitudinal directed fibers and result in further reduction in longitudinal strain [9]. Furthermore, data suggest that GLS is able to diagnose subclinical LV dysfunction [10] and to predict the prognosis of post-MI mortality independently of LVEF [11].

Natriuretic peptides (NPs) are secreted by cardiomyocytes as a result of pressure or volume overload. Although several forms of NPs have been identified, N-Terminal pro-B-type natriuretic peptide (NT-proBNP) is routinely used for diagnosis of HF, and it plays an important role in predicting outcomes and in monitoring therapeutic effectiveness. The mechanism of increasing NT-proBNP in post-MI patients with asymptomatic LV dysfunction remains unclear. Myocardial ischemia is a powerful stimulus, and, independently of changes in LVEF, it was demonstrated to induce release of NT-proBNP [12]. Previous studies described that a high concentration of NT- proBNP is associated with poor prognosis after acute coronary syndrome [13]. Recently, there has been increased interest in using plasma NT-proBNP to distinguish clinically asymptomatic patients at risk of future cardiac events.

Several studies have examined relationships between GLS and NPs, but they have mainly assessed chronic HF and cardiotoxicity. However, the link between myocardial global longitudinal deformation and neuro-hormonal activation in patients with asymptomatic LV dysfunction after AMI is poorly understood. Therefore, the aim of this study was to investigate the association between GLS and plasma NT-proBNP for predicting LV performance in asymptomatic patients post AMI.

Material and methods

Study design and patient population

We prospectively included 100 patients with diagnosis of AMI without HF. Clinical signs and symptoms and followed for 6 mos. Two participants were excluded due to incidence of acute HF during hospitalization. Thus, the study analysis was based on 98 patients. All the patients were admitted to the Coronary Care Unit of the National Cardiovascular Center at The Third State Central Hospital of Mongolia. AMI was diagnosed if a patient had acute myocardial ischemia related symptoms, dynamic electrocardiographic changes. i.e., ST-segment changes or newly discovered pathological Q wave, increased cardiac troponin, and imaging evidence of newly discovered loss of viable myocardium or regional wall motion abnormalities associated with ischemic etiology [14]. Exclusion criteria were patients with previous diagnosis and clinical symptoms of HF, age >75 years, confirmed atrial fibrillation, valvular heart disease, congenital heart disease, cardiomyopathy, kidney dysfunction, pulmonary hypertension, chronic obstructive pulmonary disease, liver cirrhosis, or anemia, since these conditions are associated with increased NT-proBNP [15].

The study was approved by the Ethics Committee of the Mongolian National University of Medical Sciences and by the Biomedical Ethics Committee of Ministry of Health of Mongolia. All participants provided written informed consent.

Demographic variables, signs and symptoms, initial hemodynamic findings (heart rate and blood pressure), cardiac risk factors, co-morbidities, past medical history and the findings of initial investigations such as blood test results, ECG data were collected from inpatient records. HF related signs and symptoms were re-obtained prior to discharge. All participants underwent immediate primary percutaneous coronary intervention (PCI). Findings in relation to coronary angiography, including the culprit lesion, number of diseased vessels, and post PCI final TIMI (Thrombolysis In Myocardial Infarction) flow grade were also obtained. Medical treatment data were collected before discharge and during follow-up visits

NT-proBNP assay

Blood samples for NT-proBNP were obtained from all participants at admission. These samples are designated as "baseline samples." Samples obtained before discharge and within 3–5 days after admission are designated as "discharge samples", and samples obtained 6 mos after discharge are designated as "6-mo samples". Blood samples were collected into tubes containing EDTA. The blood samples were stored at 4°C and centrifuged within 24 hrs. The resulting plasma was immediately frozen and stored at -80°C. NT-proBNP was measured with an immunoassay analyzer (FIA8000, Getein Bio Medical Inc, China).

Echocardiography

Echocardiographic images were obtained with a Philips iE33 Ultrasound system. Baseline echocardiography was

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performed at admission, and follow-up echocardiography was performed after 6 mos. The biplane Simpson's method was used for assessing LVEF. Two-dimensional speckle tracking measurements of GLS were performed in the two, three, and four chamber apical views. At end systole, the endocardial border was automatically traced and identified as a region of interest (ROI). The investigator visually assessed the detected ROI and, if necessary, manually adjusted the ROI to ensure that the speckles were tracked correctly. If the tracking covered the entire cardiac wall from the endocardium, it was considered adequate. A normal GLS was considered to be an absolute value of ≥16% [16]. All echocardiographic analysis were performed by a single, experienced operator.

Statistical analysis

All statistical analyses were performed using Statistical Package for the Social Sciences SPSS version 21.0. All continuous variables were tested for normality. Normally distributed data are presented as mean±standard deviation, and non-normally distributed data are presented as median (first quartile, third quartile). According to the baseline GLS, patients were divided into two groups. Baseline and 6 mos clinical characteristics were compared between the two groups using an independent t test or a Mann-Whitney U test for continuous variables and a chi-square test or a Fisher's exact test for categorical variables. Baseline and follow-up values were compared within groups using a paired t test or a Wilcoxon test for continuous variables. Correlations between

Table 1. Baseline clinical characteristics for the AMI participants stratified according to GLS

Characteristic	All patients (n=98)	Patients according to baseline GLS		p value
		Less than 16% (n=60)	Greater than 16% (n=38)	
Age, yrs	56.0±9.3	56.6±8.8	55.0±10.0	0.405
Sex, men	80 (81.6%)	47 (78.3%)	33 (86.8%)	0.289
BMI, kg/m^2	27.74±4.53	27.23±4.50	28.54±4.52	0.177
GRACE score	101.92±17.14	106.44±16.27	95.26±16.40	0.003*
Time from onset to hospital admission				
Less than 12 hr	43 (43.9%)	25 (41.6%)	18 (47.4%)	0.612
Greater than 12 hr	55 (56.1%)	35 (58.4%)	20 (52.6%)	
Co-morbidities and risk factors				
Smoking	69 (70.4%)	39 (65%)	30 (78.9%)	0.105
HTN	49 (50%)	29 (48.3%)	20 (52.6%)	0.492
DM	18 (18.3%)	13 (21.7%)	5 (13.2%)	0.405
Family history	21 (21.4%)	13 (21.7%)	8 (21.1%)	0.983
Previous MI history	12 (12.2%)	7 (11.6%)	5 (13.2%)	0.743
MI type				
STEMI	86 (87.8%)	54 (90%)	32 (84.2%)	0.123
NSTEMI	12 (12.2%)	6 (10%)	6 (15.8%)	
Number of diseased vessels				
Multi-vessel	30 (30.6%)	18 (30%)	12 (31.6%)	0.902
Single vessel	68 (69.4%)	42 (70%)	26 (68.4%)	
Physical findings				
HR, beat/min	78.35±15.33	77.06±16.68	79.23±24.12	0.604
SBP, mmHg	125.88±23.26	122.15±23.44	132.42±21.92	0.047*
DBP, mmHg	80.36±16.35	77.65±15.77	84.36±16.81	0.066
Laboratory findings				
TnI, ng/ml	3070.4 (311.57, 18527.20)	6705.75 (549.70, 39951.52)	1328.50 (132.35, 10885.20)	0.035[a]
Scr, umol/l	74.10±22.06	74.75±20.87	73.71±24.12	0.838
Medications at discharge				
Antiplatelet, %	98 (100%)	60 (100%)	38 (100%)	1
ACEI/ARB, %	96 (97.9%)	59 (98.3%)	37 (97.4%)	0.765
Beta-blockers, %	81 (82.6%)	48 (80%)	33 (86.8%)	0.331
Statin, %	97 (98.9%)	60 (100%)	37 (97.4%)	0.398

* p calculated using independent t test; [a]p value calculated using Mann–Whitney U test; p <0.05 was considered statistically significant.
BMI, body mass index; GRACE score, Global Registry of Acute Coronary Events score; HTN, hypertension; DM, diabetes mellitus; MI, myocardial infarction; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non ST-segment elevation myocardial infarction; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; TnI, troponin I; Scr, serum creatinine; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers. Data are mean±standard deviation, median (first quartile, third quartile), or number (%).

echocardiographic parameters and NT-proBNP were assessed by the Spearman's correlation coefficient. Receiver operating characteristic (ROC) curves and area under the curves (AUC) were analyzed for prediction of LV dysfunction. Statistical significance was defined as a p value <0.05.

Results

Of all 98 participants, 80 (81.6%) were males, and the mean age was 56.0±9.3 yrs. Baseline echocardiography showed that 82 (83.7%) participants had mid-range or preserved LVEF≥40%, and only 16 (16.3%) had LVEF <40%. While the majority of participants (60, 61.2%) had abnormal GLS (GLS<16%), the remainder (38, 38.8%) had normal or borderline GLS (GLS ≥16%). The basic characteristics of the participants with normal GLS group (86.8% male, mean age 55.0±10.05 years) and abnormal GLS group (78.3% male, mean age 56.6±8.8 yrs) are summarized in Table 1.

As described in Table 1, there were no significant differences in age, gender, the time from onset to hospital admission, presence of co-morbid risk factors, type of MI, kidney function, heart rate, prescribed medication and number of vessels involved in patients of the two groups. In contrast, significant differences in troponin I concentration, Global Registry of Acute Coronary Events (GRACE) Score, and systolic blood pressure were observed. Patients with abnormal GLS had a higher GRACE score, higher troponin I and lower systolic blood pressure.

Table 2 lists initial and 6-mo follow-up echocardiographic parameters, as well as values of NT-proBNP at different time points. There were significant differences in baseline echocardiographic LV systolic and diastolic function. Compared with the normal GLS group, patients with abnormal GLS had lower mean LVEF and decreased LV diastolic function, as indicated by higher average E/e' and lower mean e'. However, at 6-mo follow-up, only LV systolic function variables remained significantly different between the two groups. In comparison to baseline, there was a significant improvement of GLS in the abnormal GLS group at 6-mo follow-up (p=0.04). At 6-mo follow-up, in the abnormal GLS group, GLS values were improved in 33 (56.9%) participants, were further decreased in 5 (8.9%) participants, and remained unchanged in 20 (34.5%) participants. In the normal GLS group, the majority of the participants' GLS values remained in the normal range, but 11 (28.9%) participants had impaired GLS. During the 6-mo follow-up period, adverse events developed in 14 (14.3%) of 98 participants, and the incidence was significantly higher

Table 2. Initial and follow-up echocardiographic findings, plasma NT-proBNP concentration and incidence of complications

Characteristic	All patients (n=98)	Patients according to baseline GLS		p value
		Less than 16% (n=60)	Greater than 16% (n=38)	
Baseline echocardiographic values				
GLS, %	14.58±3.14	12.64±2.24	17.63±1.51	<0.001*
LVEF, %	49.46±6.73	46.44±5.82	54.03±5.32	<0.001*
Average E/e'	12.26±4.00	13.50±4.50	10.43±2.1	<0.001*
Mean e'	7.19±1.88	6.70±1.34	8.1±2.37	0.002*
6 mo echocardiographic values				
GLS, %	15.41±3.55	14.61±3.60[b]	17.05±2.89	0.013*
LVEF, %	51.02±8.42	49.08±8.64	54.16±7.20	0.043*
Mean E/e'	11.15±3.50	11.64±3.79	10.28±2.81	0.250
Mean e'	7.6±2.37	6.69±2.12	8.88±2.47	0.060
NT-proBNP concentrations				
Baseline NT-proBNP, pg/ml	971.50 (258, 3059.25)	1736.50 (562, 3451.50)	391 (100, 1863.50)	0.002a
Discharge NT-proBNP, pg/ml	1052 (359, 2313)	1450 (543, 2821.50)	513.50 (230.50, 1220.75)	<0.001a
6 mo NT-proBNP, pg/ml	100 (100, 313)	126.50 (100, 479.25)[b]	100 (100, 252)[b]	0.244
Incidences of complications during follow-up				
Overall	14 (14.3%)	12 (20%)	2 (5.3%)	0.042*
Hospitalization due to heart failure	4 (4.1%)	4 (6.67%)	–	0.155
Hospitalization due to unstable angina	3 (3.1%)	2 (3.33%)	1 (2.63%)	0.844
Recurrent MI	5 (5.1%)	4 (6.67%)	1 (2.63%)	0.646
Cardiovascular mortality	2 (2%)	2 (3.33%)	–	0.520

* p calculated using independent t test; [a]p calculated using Mann–Whitney U test; [b]p between initial and follow-up parameters calculated with either paired t test or Wilcoxon test. p <0.05 was considered statistically significant. GLS, global longitudinal strain; LVEF, left ventricular ejection fraction; E/e', ratio between peak early mitral inflow velocity and early diastolic mitral annular velocity; mean e', mean early diastolic mitral annular velocity; NT-proBNP, N-Terminal pro-B-type natriuretic peptide; MI, myocardial infarction.
Data are mean±standard deviation, median (first quartile, third quartile), or number (%).

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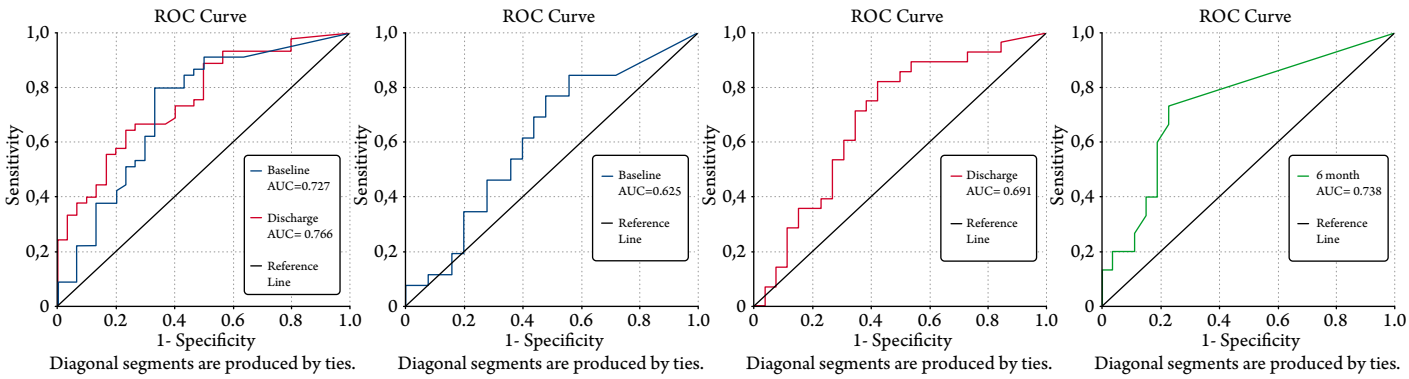
Figure 1. ROCs for relationships between NT-proBNP concentrations at different time points and baseline, follow- up GLS



A) ROC of baseline and discharge NT-proBNP concentrations to predict baseline GLS abnormality.
ROC curves of baseline (B), discharge (C) and 6 mo (D) NT-proBNP concentrations to predict 6-mo GLS abnormality.

Table 3. Correlation between NT-proBNP concentrations and LV systolic function variables

Variable	Patients according to baseline GLS			
	Less than 16% (n =60)		Greater than 16% (n =38)	
	r	p value	r	p value
Baseline NT-proBNP concentration				
Baseline LVEF (%)	–0.314*	0.038	0.289	0.093
6 mo LVEF (%)	–0.062	0.779	0.337	0.202
Baseline GLS (%)	–0.464*	0.001	0.052	0.763
6 mo GLS (%)	–0.102	0.580	0.108	0.679
Discharge NT-proBNP concentration				
Baseline LVEF (%)	–0.344*	0.012	–0.280	0.127
6 mo LVEF (%)	–0.297	0.132	–0.166	0.540
Baseline GLS (%)	–0.505*	<0.001	0.073	0.693
6 mo GLS (%)	–0.310*	0.047	0.123	0.649
6 mo NT-proBNP concentration				
6 mo LVEF (%)	–0.671*	<0.001	–0.183	0.532
6 mo GLS (%)	–0.613*	0.001	0.048	0.864

* p value is calculated using Spearman correlation and considered statistical significant at <0.05.
NT-proBNP, N-Terminal pro-B-type natriuretic peptide; LVEF, left ventricular ejection fraction; GLS, global longitudinal strain.

in the abnormal GLS group. Compared with the normal GLS group, NT-proBNP was higher in the abnormal GLS group. Baseline and discharge NT-proBNP of the two groups differed significantly (p<0.05), but there was no difference in NT-proBNP at 6 mos.

To determine whether the early NT-proBNP assessment correlated with the future changes in LV systolic function, we examined the relationship between baseline, discharge, and 6-mo NT-proBNP with baseline and 6-mo follow-up echocardiographic parameters. Table 3 shows that in the normal GLS group, NT-proBNP did not significantly correlate with echocardiographic variables at any time point. In the abnormal GLS group, baseline LV systolic function variables had a weak, negative correlation with baseline NT-proBNP and a moderate, negative correlation with discharge NT-proBNP. Discharge NT-proBNP also had a significant correlation with the 6-mo follow-up GLS, as did 6-mo NT-proBNP with follow-up LV systolic function variables.

To gain further insight into the association between NT-proBNP concentration and GLS at different time points for prediction of LV dysfunction, we generated receiver operating characteristic curves (Figure 1). For determining the effect of baseline GLS abnormality, ROC curve analysis AUC for baseline (Figure 1A) and discharge (Figure 1A) NT-proBNP concentrations were 0.73 (95% CI 0.60–0.85, p=0.001) and 0.77 (95% CI 0.66–0.87, p<0.001) respectively.

Regarding the early prediction of follow-up GLS abnormality, the area under the ROC curve for discharge (Figure 1C) NT-proBNP concentration was significantly higher (0.70 (95% CI 0.55–0.84), p=0.016) compared to baseline NT-proBNP concentration (0.62 (95% CI 0.47–0.78), p=0.122). Whereas ROC curve analysis for 6-mo (Figure 1D) NT-proBNP concentration was 0.74 (95% CI: 0.57–0.90; p=0.012). The optimum cut-off value of discharge NT-pro-BNP was 688.5 pg/ml, with 72.4%

sensitivity and 65.4% specificity to predict 6-mo GLS abnormality following AMI.

Discussion

In this study, we investigated association between GLS and plasma NT-proBNP concentrations for predicting LV dysfunction in post-AMI asymptomatic patients.

Baseline echocardiographic measurements of our study population showed that among patients with abnormal GLS, as many as 91.6% (n=54) presented with mid-range and preserved LVEF. Prior studies have reported that two thirds of patients diagnosed with AMI have preserved LVEF [17]. GLS is more prone to change early, so it has an advantage in evaluation of subclinical LV dysfunction in a variety of cardiac disorders, including MI [18, 19]. At 6-mo follow-up, there was a significant improvement in LV function as assessed by GLS in patients with initial impairment. However, compared with the normal GLS group, this group of participants had greater incidence of adverse events after discharge. As described in previous studies, GLS is a more sensitive predictor of myocardial functional recovery [20], LV remodeling [21], and major adverse events [22] in post-AMI patients. Additionally, long and short term follow-up studies have noted that following PCI, persistently abnormal or decreased GLS compared to the initial GLS presentation is associated with a higher risk of complications [23, 24].

Our study showed that plasma NT-proBNP concentrations were significantly higher in patients with abnormal GLS in the early stages of AMI. Both experimental animal and clinical investigations observed immediate and rapid secretion of NP during myocardial hypoxia and ischemia. As NT-proBNP is released immediately after myocardial infarction, peak concentrations are achieved within 24 to 36 hrs after onset and will decrease significantly following successful reperfusion [12, 25]. Our results are consistent with this finding. In this study we investigated relationships between NT-proBNP concentrations and LV systolic function at different time points. Results showed that assessment of NT-proBNP at acute and subacute phases of AMI had a significantly stronger relationship to GLS compared to LVEF. Positive correlations were found between baseline and follow-up GLS with discharge NT-proBNP concentration. This means that discharge NT-proBNP concentration could be a reliable predictor of baseline and follow-up LV dysfunction.

Very few studies have demonstrated a relationship between GLS and NP in AMI. Ersbøll et al. described that in post-MI patients, impaired GLS had a strong correlation to plasma NT-proBNP concentration as well as to the risk of HF during hospitalization [26]. Thus, in post-MI asymptomatic patients, LV global longitudinal strain and plasma NT-proBNP concentration could reflect an intrinsic abnormality of LV function. We found that baseline and discharge NT-proBNP concentrations could equally predict baseline GLS abnormality. Whereas for prediction of LV dysfunction at 6 mos, only discharge NT-proBNP concentration had predictive value. Prognostic significance of plasma NT-proBNP at different time points in AMI have been described previously, but still there is concern about optimal timing of NT-proBNP measurement for better prediction of clinical outcome. Heeschen C et al. reported that in acute coronary syndrome, a higher NT-proBNP concentrations after 72 hrs of onset was linked to short term adverse prognosis [13]. Also Eurlings LW et al. [27] reported that NT-proBNP concentrations at discharge or soon after discharge were associated with the outcome. Our findings are similar to these findings. Instead of admission NT-proBNP concentration, NT-proBNP at discharge was a better predictor of 6-mo LV dysfunction. This indicates that early NT-proBNP concentration is more related to myocardial necrosis-induced release of natriuretic peptide, but in the subacute phase, release of the NT-proBNP corresponds to infarct expansion and evolution of LV systolic dysfunction following AMI.

In summary, we observed that combination of initial LV GLS and NT-proBNP concentration at pre-discharge improves personalized stratification and identification of patients needing intensive monitoring and early optimization of treatment to prevent future cardiovascular complications.

Limitations of this study includes the small sample size. Thus, further studies are required for clarification. Also, pre-discharge NT-proBNP measurements were not conducted at one standardized time point. Blood sampling was performed 3–5 days after admission, so we cannot fully exclude a possible influence of the time differences in blood sampling. Furthermore, 12% of the participants had previous MI history, which could have impacted the evolution of LV systolic function.

Conclusion

The main finding of this study is that impaired LV GLS is associated with elevated plasma concentrations of NT-proBNP in post AMI patients. Pre-discharge NT-proBNP concentration combined with impaired initial GLS could predict worsening LV systolic function over time in asymptomatic post-AMI patients.

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СЕРДЦЕ НУЖДАЕТСЯ

В ДОПОЛНИТЕЛЬНОЙ ЗАЩИТЕ ПОСЛЕ ЭПИЗОДА ДЕКОМПЕНСАЦИИ СН[1]

Пациенты особенно уязвимы в ранний период после стабилизации состояния[2,*]

- Ежегодно у **~30% пациентов** с симптоматической хронической СН развиваются эпизоды **декомпенсации СН**[3]
- В России **повторные госпитализации** после эпизода острой декомпенсации СН регистрируются **у 31% пациентов в течение 1 месяца, а у 63,4% - на протяжении первого года**[4]
- **Смертность** пациентов с СНнФВ **в течение 3 лет** после выписки из стационара составляет **от 20% до 48%**[5]

Патофизиологические механизмы развития СН и возможные точки приложения лекарственных препаратов, улучшающих прогноз:[6]

Угнетение сигнального пути NO-рГЦ-цГМФ имеет важное значение в прогрессировании заболевания[7,8]

NO - оксид азота; ЛЖ - левый желудочек; РААС - ренин-ангиотензин-альдостероновая система; рГЦ - растворимая гуанилатциклаза; СН - сердечная недостаточность; СНнФВ - сердечная недостаточность со сниженной фракцией выброса; цГМФ - циклический гуанозинмонофосфат

* На основании анализа серии пациентов с хронической СН после эпизода острой декомпенсации СН: в первые 6 месяцев наблюдались максимальные относительные различия показателей общей и сердечно-сосудистой смертности между пациентами (n=510), наблюдавшимися в специализированном центре лечения СН, и пациентами (n=432), наблюдавшимися в амбулаторно-поликлинических учреждениях по месту жительства.

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МАТЕРИАЛ ПРЕДНАЗНАЧЕН ДЛЯ СПЕЦИАЛИСТОВ ЗДРАВООХРАНЕНИЯ

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