



ORIGINAL RESEARCH ARTICLE

## Improving predictive value of transient ischemic dilation ratio after correction based on the left ventricular mass

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### ABSTRACT

**Introduction:** For the interpretation of myocardial perfusion imaging, considering non-perfusion variables, such as transient ischemic dilation (TID), are also important in diagnostic and prognostic evaluation of a patient. TID has a relatively high false positive and false negative results, which reduces its diagnostic and prognostic values. In this study, we aimed to compare the accuracy of TID after normalization based on changes in LV wall mass.

**Methods:** Patients referred for dipyridamole myocardial perfusion imaging single-photon emission computed tomography (MPI/SPECT), one to two year prior to the study with TID ratio >1 were enrolled. Patients with any previous history of revascularization and structural heart disease were excluded. Follow-up was done by phone call. The occurrence of cardiac death, myocardial infarction, revascularization or evidence of abnormal angiography during one year after MPI was considered positive for short-term cardiac events. The corrected TID (cTID) was calculated by the following formula:  $cTID = TID / (Wall_{stress} / Wall_{rest})$ .

**Results:** Among 196 participants, 30 (15%) had cardiovascular events during the follow-up. The areas under the receiver operating characteristic (ROC) curve for the short-term prognosis of the cardiac events were 0.57 and 0.50 for cTID and TID with a p-value of 0.17 and 0.92, respectively. Considering the best cut-off points that were achieved by ROC curves, cTID showed significant odds ratio (OR: 2.53) for prediction of short-term cardiac events, while respecting, TID failed to be statistically significant.

**Conclusion:** Making correction on TID using LV wall volumes can improve short-term prognostic value of this variable.

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## INTRODUCTION

Single-photon emission computed tomography (SPECT) myocardial perfusion imaging [1] is a noninvasive method for the evaluation of coronary artery diseases (CAD). In addition to the qualitative and quantitative assessment of perfusion findings, there are several non-perfusion variables, indicating different aspect of CAD consequences on the heart [2-4]. These include functional parameters, lung-to-heart ratio, right ventricular visualization and transient ischemic dilation (TID) of left ventricle (LV). These parameters can further enhance the diagnostic and prognostic accuracy of MPI/SPECT [2, 5]. TID, defined as increased ratio of LV cavity size at stress to LV cavity size at rest, is considered as a diagnostic indicator of severe and extensive disease and in direct association with worse prognosis [6]. However, the diagnostic and prognostic values of TID, especially in normal scans incline to be questionable, ranging from no value in some researches to beneficial in others [7, 8]. The prognostic studies have also reported different predictive ability for TID in patients with normal and abnormal scans [6].

Although the exact mechanism of TID is unclear, the more accepted underlying pathophysiology, especially following pharmacologic stress, is diffuse sub-endocardial ischemia which causes just an apparently dilated LV cavity with no real cavity enlargement [1, 9, 10]. Another mechanism is true LV cavity dilation in stress phase due to LV stunning, a phenomenon that is more commonly seen after exercise stress [1]. It has also been suggested that in some patients both mechanisms are involved [10]. In addition to these pathologic mechanisms of a true TID, there are many clinically proven and other unknown artifactual and pathologic causes of false higher TID ratio that can adversely impact the value of TID ratio as a useful diagnostic and prognostic marker [11, 12]. These artifactual causes are considered as the most important factor that limit the value of TID, especially in normal scans in which the prevalence of abnormal TID is relatively low [13]. The artifactual causes including different heart rate, zoom factor, selection of slices, radiotracer dose or distance from the detector and sometimes unknown factors, which may result in enlargement of the whole LV including both LV cavity and myocardial wall indices, producing a false positive TID, whereas pathologic mechanisms with sub-endocardial ischemia are believed to cause apparent LV cavity enlargement with thinner wall [11] leading to decrease in overall LV myocardial mass. Regarding this theoretical difference, we aimed to normalize TID ratio for exclusion of false positive TID to the changes in LV mass, provided by

most myocardial perfusion quantitative software packages, and then compare it to conventional TID ratio in practice for prognostic evaluation.

## METHODS

### *Study population*

This study was done prospectively in two centers. All patients who underwent MPI/SPECT in a 6-months period in these two centers were evaluated. The Ethics Committee of Shiraz University of Medical Sciences approved the study's design (IR.SUMS.MED.REC.1396.s171). The inclusion criteria consisted of patients with TID ratio >1 who underwent dipyridamole stress test without any past history of CAD and acceptable quality of MPI/SPECT. Patients with history of dialysis, structural heart disease or those with questionable history or incomplete follow-up data were excluded.

### *MPI/SPECT*

All patients underwent two-day MPI protocol with dipyridamole stress test on the first day and rest phase on the second day. Routinely in the two centers, 0.56 mg/kg of dipyridamole is intravenously infused during 4 minutes when pharmacologic stress test with vasodilators is indicated. Two minutes after completion of dipyridamole infusion, 555-925 MBq (15-25 mCi) <sup>99m</sup>Tc-sestamibi is injected with the same dose of activity used for the rest phase. SPECT acquisition is performed 60-90 minutes after radiotracer injection in both phases with a dual-head gamma camera dedicated to cardiac imaging (same commercial brand in both centers). A 180° arch, from right anterior oblique view to left posterior oblique view, containing sixty 30-second projections is used. Then, raw projection data reconstructed by ordered subsets expectation maximization (OSEM) 3D algorithm (subset:4, order:8) with post-filtering (Butterworth; cutoff: 0.5, order: 8). Quantitative and semi-quantitative data including summed stress score (SSS), summed rest score (SRS), summed difference score (SDS) and TID as well as total perfusion deficit (TPD), extent, cavity volume and wall mass at both phases are derived by Quantitative Perfusion SPECT(QPS)/ Quantitative Gated SPECT(QGS) software.

MPI/SPECT images were interpreted by two nuclear medicine specialists who were blinded to the patients' follow-up results. Interpretation was based on 4 categories: normal perfusion, mild, moderate and severe ischemia. All images were reviewed on a monitor. In case of discordant interpretations, a third specialist with the same level of expertise, reported the scan. The two similar results were considered as the final result. In case of completely

discordance, the case was omitted from related analyses.

#### Calculation of TID and corrected TID (cTID) ratios

TID was automatically derived from QPS software, which calculates TID as ratio of stress cavity volume to rest cavity volume in summed perfusion data [14]. We assumed that the relative enlargement of LV cavity due to enlargement of whole LV is equal to relative enlargement of other parts of LV including the LV mass. Hence, if the TID ratio, which is the ratio of LV cavity volumes at stress and rest was greater than the ratio of other parts of LV myocardium (LV mass) at both phases, this difference is due to only LV cavity dilation. In other words, if the ratio of TID/ (stress LV mass/ rest LV mass) is greater than 1, we can conclude that LV cavity at stress is greater than the rest phase regardless of how much other technical factors affect the whole size of LV. Consequently, we defined this new ratio as corrected TID (cTID).

#### Follow-up

All patients were followed-up via phone call after 1 year with questions about evidence of any hard (cardiac death or non-fatal myocardial infarction) or soft (revascularization or unstable angina) cardiac events. If the incidence of event was reported by the patients or their relatives during the first year after MPI, further documentation were asked to confirm

and categorize the event. Patients who were inaccessible, unreliable or refused further therapy or management were excluded from the study.

#### Statistical analysis

For descriptive and analytical analysis, SPSS software version 16 and for the comparison of the receiver operating characteristic (ROC) curves Medcalc software version 15/8 were used. Data are represented as mean± standard deviation for continuous quantitative variables and number (%) for frequencies. For the comparison of event and nonevent groups chi square and independent samples t-test were utilized. We used ROC curve to estimate a cut-off point for TID and cTID for predicting cardiac events. For the comparison of prognostic value of TID and cTID for the prediction of 1-year cardiac events ROC curves and odds ratio were used. P value<0.05 was considered to be statistically significant.

## RESULTS

Finally, 196 patients were included (136 from Namazi and 60 patients from Alzahra Hospital). Basic clinical characteristics of patients consisting of mean age and frequency of diabetes mellitus, hypertension, hyperlipidemia, smoking, positive family history of CAD and cardiac symptoms are shown in Table 1.

**Table 1.** Baseline clinical characteristics in event and non-event groups

Clinical characteristics	Event (n = 30)	Non-event (n = 166)	P Value
Age	58.5 ± 10.7	56.7 ± 10.7	0.408
Gender	66.6	28.3	<b>&lt;0.001</b>
Diabetes mellitus	20	22.2	0.375
Hypertension	76.6	63.2	0.155
Hyperlipidemia	46.6	42.7	0.692
Smoking	20	19.2	0.951
Family history	10	22.2	0.115
Presence of typical angina	16.6	10.2	0.252

Dichotomous variables: frequency (%) and Continuous variables: mean ± SD.  
P values in bold represent statistical significance.

It is clear that only gender type is significantly different between event and non-event groups (p-value < 0.001). After 1-year follow-up, 150 (76.5%) patients experienced no event with no need for further diagnostic work up, and 16 (8%) patients had normal or unremarkable angiographic results. These patients were considered as non-event group. Other 30 (15.3%) patients who made the event group including 2 (1%) patients with cardiac death, 2 (1%) patients with non-fatal MI, 17 (8.6%) patients with revascularization (7 CABG and 10 PCI) and 9 (4.5%) patients with abnormal angiographic results without revascularization. Comparison of

mean value of quantitative perfusion parameters between the two event and non-event groups are shown in Table 2. Except for TID and cTID, all other parameters including SSS, SRS, SDS, stress and rest extent, TPDs, TPD<sub>r</sub> and ΔTPD revealed statistically significant difference (Table 2).

In visual interpretation, 165 patients were categorized in normal perfusion, 13 in mild ischemia, 9 in moderate ischemia and 9 in severe ischemia groups. Total of 4 cases categories were different in two interpretations that were judged based on third specialist opinion. Agreement of the two interpretations was good (kappa value = 0.935).

**Table 2.** Comparison of mean value of quantitative perfusion parameters

	Event (n = 30)	non-event (n = 166)	P Value
cTID	1.10 ± 0.11	1.07 ± 0.06	0.179
TID	1.16 ± 0.19	1.13 ± 0.10	0.923
Stress extent	12.90 ± 13.81	4.39 ± 6.24	<b>&lt;0.001</b>
Rest extent	0.43 ± 0.50	0.10 ± 0.37	<b>0.002</b>
SSS	10.5 ± 11.1	4.1 ± 5.0	<b>&lt;0.001</b>
SRS	3.9 ± 5.5	1.6 ± 3.6	<b>0.001</b>
SDS	6.7 ± 6.5	2.6 ± 2.9	<b>0.003</b>
TPDs	10.6 ± 10.8	4.3 ± 5.2	<b>0.004</b>
TPDr	3.0 ± 5.6	1.1 ± 3.6	<b>0.016</b>
ΔTPD	7.6 ± 7.6	3.2 ± 2.9	<b>&lt;0.001</b>

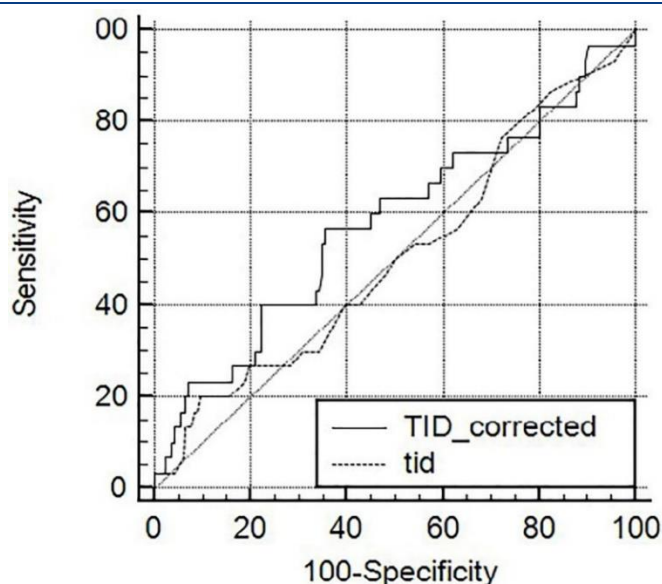
Continuous variables: mean ± SD.

P values in bold represent statistical significance.

cTID: Corrected transient ischemic dilation, TID: Transient ischemic dilation, SSS: Summed stress score, SRS: Summed rest score, SDS: Summed difference score, TPDs: Stress total perfusion deficit, TPDr: Rest total perfusion deficit, ΔTPD: Delta total perfusion deficit (TPDs-TPDr).

After TID correction according to myocardial volume changes, the new TID ratio decreased in 141 patients and increased in 36 patients with no change in 19 patients. In the ROC curve analysis,

area under the curve (AUC) was calculated as 0.57 for cTID and 0.50 for TID (p-value = 0.92 for TID and p-value = 0.17 for cTID). ROC curve graph of both variables are shown in Figure 1.



**Fig 1.** Comparison of TID and cTID ROC curves for prediction of cardiac events at 1 year

Comparing the ROC curves of the two variables indicates that the difference in AUCs is not significant (p-value = 0.13). Prognostic cut-off was found to be 1.14 for TID and 1.11 for cTID. Accepting these cut-offs prevalence was calculated as 40% for TID and 25% for cTID in our sample, which includes only patients with TID>1. Applying this cut-off, TID shows sensitivity and specificity of 40% and 60%, respectively. Using prognostic cut-off sensitivity and specificity of cTID reached 40% and 78%, respectively. Considering these cut-off values, 6 patients with normal TID were re-categorized as abnormal by cTID. Three out of 6 patients (50%) experienced

an event in their follow-up. On the other hand, in patients with abnormal TID, 29 patients were also re-categorized as normal, of whom 25 patients (86%) had no cardiac events in their follow-up. Further analysis revealed that cTID could reduce the number of false positive results from 55 to 37 patients without any change in number of true positive and false negative results. Unadjusted risk determination of cardiac events was done using these cut-off values. Cardiac death and nonfatal MI, did not contain the minimum count of cases to undergo statistical analysis. Positive TID shows no incremental value to predict any categories of adverse cardiac event (Table 3).

**Table 3.** Unadjusted risk of cardiac events for TID

	Entire study 196	TID+ 78 (40)	TID- 118 (60)	Odds ratio (95% CI)	P value
Cardiac death or MI	4 (2)	0 (0)	4 (3)	NA	0.10
Revascularization (CABG or PCI)	17 (9)	8 (10)	9 (8)	1.38 (0.51-3.75)	0.52
Cardiac death, MI, revascularization	21 (11)	8 (10)	13 (11)	0.92 (0.36-2.34)	0.86
Cardiac death, MI, revascularization, abnormal angiography	30 (15)	12 (15)	18 (15)	1.01 (0.45-2.23)	0.98

P values in bold represent statistical significance.  
Dichotomous variables: frequency (%).  
TID: Transient ischemic dilation, CI: Confidence interval, MI: Myocardial infarction, CABG: Coronary artery bypass graft, PCI: Percutaneous coronary intervention, NA: Not analyzed.

However, with newly produced variable, cTID, positive group had higher rate of revascularization (CABG or PCI) as well as composite endpoint of adverse cardiac events consisted of cardiac death, nonfatal MI,

revascularization and abnormal angiography result. Results of unadjusted risk determination of adverse cardiac events for cTID are shown in Table 4.

**Table 4.** Unadjusted risk of cardiac events for cTID

	Entire study 196	cTID+ 49 (25)	cTID- 147 (75)	Odds ratio (95% CI)	P value
Cardiac death or MI	4 (2)	1 (2)	3 (2)	1.00 (0.10-9.84)	1.00
Revascularization (CABG or PCI)	17 (9)	8 (16)	9 (6)	2.99 (1.08-8.24)	<b>0.02</b>
Cardiac death, MI, revascularization	21 (11)	9 (18)	12 (8)	2.53 (0.995-6.43)	0.04
Cardiac death, MI, revascularization, abnormal angiography	30 (15)	12 (24)	18 (12)	2.32 (1.02-5.26)	<b>0.03</b>

P values in bold represent statistical significance.  
Dichotomous variables: frequency (%).  
cTID: Corrected transient ischemic dilation, CI: Confidence interval, MI: Myocardial infarction, CABG: Coronary artery bypass graft, PCI: Percutaneous coronary intervention, NA: Not analyzed.

## DISCUSSION

TID is suggested to be a predictor of poor short-time outcomes and extensive CAD [15], though there are some controversies regarding its value in different settings [6, 7, 16]. The current study revealed that modification of TID ratio according to the most accepted underlying pathophysiologic mechanism, i.e. diffuse sub-endocardial ischemia, can potentially strengthen the prognostic ability of this parameter, most likely by omitting the interfering effect of technical factors. Earlier, when both epicardial and endocardial TID were introduced as a diagnostic and prognostic ancillary marker in the planar and MPI/SPECT, the exact pathological mechanism was unknown [17]. With further investigation, knowledge about possible underlying mechanism became available [11]. Currently, the most accepted theory to explain the underlying cause of TID, especially in case of pharmacologic stress, is diffuse sub-endocardial ischemia resulting in diminished sub-endocardial tracer uptake contributing to an appearance of LV dilation with thinner wall [11]. Post-stress LV stunning is also considered as another mechanism for increased TID, especially when exercise stress test is performed [14, 18], resulting in true LV cavity dilation. However, some authors questioned the value of TID, particularly

in otherwise normal MPI/SPECT in whom the prevalence of TID and cardiac events is low. Since TID depends on post-stress and rest LV volumes, it can be stated that any factors that can affect these two parameters can influence TID ratio [19]. Indeed, diagnostic and prognostic value of TID suffer from multiple unwanted confounders amongst those are factors related to the patient, some in association with camera and acquisition, and others related to the radiotracer used [20]. However, it is critical to differentiate CAD-related TID from other causes. Impact of these non-CAD-related factors might be best presented in patients with normal myocardial perfusion findings in which some authors found no significant additional diagnostic or prognostic value of TID. Some authors attribute this finding to lower sensitivity of TID in whom lower prevalence of severe CAD leading to the higher rate of false positive results [13]. Thus, to keep the accuracy in an acceptable range we need to increase specificity by further exclusion of false positive cases.

Considering the fact that common underlying mechanisms of TID would not increase LV mass as much as myocardial volume, we decided to normalize the TID by the ratio of myocardial mass (volume) to find cases in whom the increase in LV mass was larger than increase in LV volume. We assumed that in these cases artificial or

technical factors, such as difference in zoom factor, selection of slices, radiotracer dose or distance from the detector in both phases and other unknown but interfering factors would be the most likely causes. The results of this study, is improved prognostic value of the TID after this volume-based modification (cTID) and deceased number of patients who categorized falsely as high-risk by TID, supported our hypothesis and further emphasis on the role of sub-endocardial ischemia as a strong explanation for TID at least in vasodilator stress.

Making correction on TID using LV wall volumes in this study showed acceptable ability to omit some confounding factors that interfere with TID accuracy in prognostic function. Using ROC curve to estimate the best cut-off point for our new parameter, cTID, for the prediction of annual cardiac event, this parameter could enhance the specificity by 18% (60% for TID and 78% for cTID) at a cut-off level of 1.11 with same level of sensitivity as compared to TID. Odds ratio is significant for composite of all adverse cardiac event including composite endpoint of cardiac death, MI, revascularization and abnormal angiography (2.32, 95%CI: 1.02-5.26). It happened while TID itself failed to predict any group of adverse outcome with either cut-off level of 1.14 (estimated from our ROC analysis; OR: 1.01, 95%CI: 0.45-2.23) or 1.19 as suggested by previous studies [18]. These results are in contrast with studies that had concluded the prognostic importance of TID in general [6] or in special conditions, such as positive history of diabetes mellitus or CAD [21]. However, a recent meta-analysis reported pooled sensitivity of 44% and pooled specificity of 88% for the detection of extensive CAD and indicated that TID is a predictor of poor outcome disease. Although our study showed that myocardial-based normalization of TID can improve its prognostic function, however, the new parameter has still some limitations to meet the expectation as an optimal prognostic marker. An important explanation could be the fact that there are also factors rather than CAD resulting in true TID, which might not have adverse outcomes as bad as CAD. Hypertension, myocardial hypertrophy and diabetes mellitus are accounted in this group [22-24]. On the other hand, the technical factors would not always lead to the same degree of increase in both LV mass and LV volume. Another reason could be the relatively small number of cases included in the study demonstrating low rate of events during the follow-up (15% event rate). The lower event rate could also be due to short follow-up. In our study patients were

followed for one year. Longer follow-up periods were used in similar studies. Follow-up period were 2.3 years in a study by Abidov et al. [15] more than 2 years in Petretta et al. [25] and 18 months in uz Zaman et al. [26] studies. The shorter the follow-up the lower the event rate. Hence, following patients for longer duration probably would result in higher event rate and more confident conclusion. Although such patient selection might not significantly influence the results of TID and cTID comparison, it could have some effect on overall accuracy of these variables. Even though we included only patients with TID>1, the rate of positive TID in our sample was low, estimated 25-38% (considering both cut-off points of 1.14 and 1.19). Some shortcomings in this study could be rectified by conducting a larger study with a larger sample size. Additionally, in our study all patients had underwent vasodilator stress with TID>1 and no proven history of CAD. This makes the results more specific for this subgroup of patients and generalization of these results to other protocols and other population requires further investigation.

## CONCLUSION

Making correction on TID using LV wall volume, improves the short term predictive value of this parameter. However, further investigations and validations are required.

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