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Prospective Study Correlating Level of Troponin I and Regional wall motion abnormality by Tissue Doppler Imaging in patients with Non-ST Elevation Myocardial Infarction.

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Abstract

Introduction: Quantification of Regional wall motion abnormality is a major unresolved issue in Cardiology. We evaluated pulsed Systolic Doppler Tissue Imaging(DTI),to quantify regional wall motion abnormality (RWMA) in Patients of Non-ST Elevation Myocardial Infarction. Our Study aimed to identify a cut of value of Troponin I at which RWMA appears by DTI in patients of NSTEMI and whether any correlation exists between the level of Troponin I and Quantified RWMA by DTI and Finally to test whether DTI can identify those patients of NSTEMI in whom there is no RWMA by visual estimation. ***Methods***: Normal Pulsed Systolic Tissue Doppler velocities at each of Myocardial segments were taken from 25 healthy subjects. Then Pulsed Systolic DTI was performed in 25 patients of NSTEMI after 24 hrs of presentation of chest pain. Velocities lower than the Normal velocities at that segment were identified as RWMA and quantified. These were then correlated with Troponin I values which were taken at admission and 12 hrs after chest pain to see whether any linear relationship exists. ***Results***: Regional wall motion abnormality (RWMA) was identified and quantified in 17(68) out of 25 patients. The cut of value of Troponin I above which RWMA could be quantified was found to be >0.5 which was statistically significant (P <0.001). 8(32%) patients in whom Troponin I value were less than 0.5 there was neither RWMA visualized by 2D echo nor Quantified by TDI. However, there were 9(53%) patients in whom Troponin I value were more than 0.5 but there was no RWMA by visual assessment but which could be identified and quantified by Pulsed Systolic TDI. Though TDI could identify and quantify RWMA, there was no correlation between Level of Troponin I and RWMA. ***Conclusions***: Pulsed TDI is a non-invasive technique by which RWMA can be quantified in patients with NSTEMI which is reliable, reproducible and accurate. Pulsed Systolic TDI helps in identifying RWMA which may otherwise be missed by visual assessment. Though Pulsed Systolic TDI identifies and quantifies RWMA there appears to be no correlation between level of Troponin I and quantified regional wall motion abnormality.

INTRODUCTION

The burden of Non-ST Elevation Myocardial Infarction is increasing globally as compared to ST Elevation Myocardial infarction.[1-9]. Troponin I detects level of Myocardial injury well below threshold required to result in Regional wall motion abnormality. Levels of Troponin at which regional wall motion abnormality appear is not well known. Detection of myocardial ischemia by visual assessment of wall motion is fraught with variability and low reproducibility. Wall motion can be quantified by Tissue Doppler imaging.[1] Tissue velocity, Strain and Strain rates are Tissue Doppler modalities to Quantify Regional wall motion abnormality. Low systolic tissue velocities correlate with Echocardiographic wall-motion abnormality.[3,10] Tissue Doppler detects more subtle degree of myocardial dysfunction than are apparent by visual analysis and can therefore be used as marker of Acute ischemia. Tissue Doppler as marker of Global LV Function have already been studied but its value in assessing regional wall motion abnormality is not well known. There is therefore need to utilize such easy and reproducible technique to identify and quantify Regional wall motion abnormality in patients with Acute coronary syndrome which is not only qualitative but also quantitative , reproducible and may therefore become a useful diagnostic tool in patients with Acute coronary syndrome.

Objectives:

1. To predict level of Troponin I at which Regional Wall motion abnormality appears by Pulsed Systolic Tissue Doppler Imaging
2. To correlate Troponin I levels in Patients with Non ST Elevation Myocardial Infarction and quantify Regional wall motion abnormality by tissue Doppler

3. To predict percentage of patients with Regional wall motion abnormality by Pulsed Systolic Tissue Doppler in patients with no Regional wall motion abnormality by visual assessment

MATERIALS AND METHODS

This study was conducted at Amrita Institute of Medical Sciences, Kochi, India, over a six-month period from January 2014 to June 2014. A total of 25 patients diagnosed with acute coronary syndrome presenting as non-ST-elevation myocardial infarction (NSTEMI) were included in the study.

Patients presenting within 24 hours of the onset of chest pain and diagnosed with NSTEMI with a positive cardiac troponin I (cTnI) result were eligible for inclusion. All enrolled patients underwent a detailed clinical evaluation, including a comprehensive cardiac history and physical examination. Serum troponin I levels were measured at admission and at 12 hours after the onset of chest pain.

All patients underwent echocardiographic assessment using pulsed systolic tissue Doppler imaging within 24 hours of presentation with chest pain. Regional myocardial function was evaluated, and regional wall motion abnormalities (RWMAs) were quantified based on the echocardiographic findings. The relationship between troponin I levels and the presence and extent of regional wall motion abnormalities was subsequently assessed.

Patients with previously diagnosed coronary artery disease were excluded from the study. Patients with NSTEMI who presented more than 24 hours after the onset of symptoms were also excluded. In addition, patients with clinical conditions known to be associated with elevated troponin I levels in the absence of acute coronary syndrome, including renal failure, sepsis, and heart failure, were excluded.

Statistical Analysis: Data was entered into Microsoft excel data sheet and was analyzed using Epi-info version 7.2.1 (CDC Atlanta) software and Jamovi (Version 2.6.26.0). Statistical analysis was performed to evaluate the relationship between serum troponin I levels and regional wall motion abnormalities. Pearson's correlation coefficient was calculated to determine the strength and direction of the association between troponin I levels and the quantitative assessment of regional wall motion abnormalities. For categorical analysis, patients were classified according to the presence or absence of abnormal regional wall motion, and Student's t-test was used to assess differences in troponin I levels between the groups. A p-value below the <0.05 considered statistically significant. The diagnostic performance of regional wall motion abnormality in relation to troponin I-based grouping was evaluated by calculating validity parameters, including sensitivity, specificity, and overall diagnostic accuracy.

RESULTS

Normal segment-specific pulsed systolic tissue Doppler imaging (TDI) velocities were determined from 25 age- and sex-matched healthy controls. In patients with non-ST-elevation myocardial infarction (NSTEMI), a systolic tissue Doppler velocity below the corresponding segment-specific normal value was considered indicative of a regional wall motion abnormality (RWMA), and the abnormality was quantified using pulsed systolic TDI.

Among the 25 patients with NSTEMI, quantifiable RWMAs were detected by pulsed systolic TDI in 17 patients (68%), whereas 8 patients (32%) had no quantifiable RWMA. The segment-wise distribution of RWMAs among the 17 affected patients was assessed; however, the numerical segment-level data were not available in the material provided and therefore are not reproduced here. All 8 patients (32%) without a quantifiable RWMA had troponin I values <0.5, whereas all 17 patients (68%) with a quantifiable RWMA had troponin I values >0.5. The difference in troponin I values between patients with and without quantifiable RWMA was statistically significant (P<0.001).

Table 1: Comparison of Troponin I Levels According to Regional Wall Motion Abnormality

RWMA by TDI	Troponin I						*P* value
	N	Mean	SD	Median	Minimum	Maximum	
Normal (no RWMA by TDI)	8	0.14	0.31	0.03	0.01	0.91	**<0.001***
Abnormal (RWMA+)	17	7.51	13.9	1.65	0.06	50	

Patients were further categorized according to troponin I concentration as having a low troponin I value (<1.0) or a high troponin I value (>1.0). Among patients in whom RWMA was identified, 7 patients had troponin I values >0.5 but <1.0. All 10 patients in the high troponin I group (>1.0) had a quantifiable RWMA. In the low troponin I group (<1.0), 7 of 15 patients (46.7%) had an abnormal RWMA and 8 (53.3%) had no quantifiable RWMA. The association between troponin I group and quantifiable RWMA was statistically significant (P=0.016).

Table 2: Association between troponin I group and quantifiable regional wall motion abnormality

Troponin I group	Abnormal RWMA, n (%)	Normal RWMA, n (%)	P value
High (>1.0)	10 (100.0)	0 (0.0)	0.016
Low (<1.0)	7 (46.7)	8 (53.3)	

Using the reported validity parameters, pulsed systolic TDI for the identification and quantification of RWMA had a sensitivity of 58.8%, specificity of 100%, positive predictive value of 100%, negative predictive value of 53.3%, and overall accuracy of 72.0%.

Table 3: Diagnostic validity parameters of pulsed systolic tissue Doppler imaging for RWMA

Validity parameter	Value
Sensitivity	58.8%
Specificity	100.0%
Positive predictive value (PPV)	100.0%
Negative predictive value (NPV)	53.3%
Accuracy	72.0%

Among the 17 patients with RWMA detected by pulsed systolic TDI, 9 (53%) had no identifiable wall motion abnormality on visual analysis of two-dimensional echocardiography, whereas 8 (47%) demonstrated RWMA by both pulsed systolic TDI and visual assessment of two-dimensional echocardiography.

Pearson's correlation analysis was performed to assess the relationship between troponin I levels and segment-specific RWMAs identified using pulsed systolic TDI. No significant correlation was reported between troponin I levels and RWMAs in the individual myocardial segments.

DISCUSSION

Tissue Doppler imaging (TDI) provides quantitative assessment of myocardial motion and has demonstrated incremental value over conventional echocardiography in several clinical settings. Nevertheless, despite the accumulating evidence supporting its utility, measurement of tissue velocities has had relatively limited routine clinical application[3]. TDI enables objective characterization of myocardial systolic function and may complement the predominantly qualitative assessment of regional wall motion performed using conventional two-dimensional (2D) echocardiography. Although cardiac magnetic resonance imaging can provide quantitative assessment of myocardial wall motion, its availability and feasibility may be limited in routine clinical practice [1].

Conventional echocardiographic assessment of regional wall motion is largely dependent on visual interpretation and may therefore be subject to intra- and interobserver variability. The addition of TDI provides a quantitative measure of myocardial motion that may improve the detection of subtle abnormalities. In particular, reductions in peak systolic myocardial velocity have been proposed as a marker of coronary artery disease (CAD), including in resting examinations and in patients with suboptimal 2D endocardial definition [5]. Experimental studies have also demonstrated reduced systolic tissue velocities in ischemic myocardial segments[5]. However, the role of pulsed systolic TDI in identifying and quantifying regional wall motion abnormalities (RWMAs) in patients presenting with non-ST-elevation myocardial infarction (NSTEMI) remains less well established.

The present study evaluated the ability of pulsed systolic TDI to detect and quantify RWMAs in patients with NSTEMI and examined their relationship with serum troponin I levels. An additional objective was to determine whether TDI could detect regional myocardial abnormalities that were not apparent on routine visual assessment by 2D echocardiography.

Among the 25 patients with NSTEMI included in the study, 17 (68%) demonstrated quantifiable RWMAs by pulsed systolic TDI, whereas 8 (32%) had no quantifiable RWMA. Troponin I levels differed significantly between these groups. Patients without a quantifiable RWMA had a mean troponin I level of 0.14, compared with 7.51 among patients with an abnormal RWMA (P<0.001). Median troponin I levels were 0.03 and 1.65, respectively. These findings demonstrate an association between higher troponin I concentrations and the presence of RWMAs detected by pulsed systolic TDI.

When patients were categorized according to troponin I level, all 10 patients (100%) in the high troponin I group (>1.0) demonstrated a quantifiable RWMA. In contrast, among the 15 patients in the low troponin I group (<1.0), 7 (46.7%) had a quantifiable RWMA and 8 (53.3%) had no quantifiable RWMA. This association was statistically significant (P=0.016). These findings suggest that RWMAs detected by TDI were more frequently observed among patients with higher troponin I concentrations.

The data also showed that all patients without a quantifiable RWMA had troponin I concentrations below 0.5, while patients with quantifiable RWMAs had values extending above this level. However, the observed data do not establish 0.5 as a definitive diagnostic threshold for the development or detection of RWMA. Notably, the reported troponin I range among

patients with RWMA extended as low as 0.06. Moreover, a formal receiver operating characteristic analysis was not reported, and the small sample size limits determination of an optimal cut-off value. Therefore, the findings are more appropriately interpreted as demonstrating an association between troponin I levels and the presence of TDI-detected RWMA rather than establishing a specific troponin I threshold.

In the reported validity analysis, pulsed systolic TDI demonstrated a sensitivity of 58.8%, specificity of 100%, positive predictive value of 100%, negative predictive value of 53.3%, and overall accuracy of 72.0% for identifying and quantifying RWMA. The high specificity and positive predictive value observed in this cohort indicate that an abnormal TDI finding was strongly associated with the reference classification used in the study. However, the moderate sensitivity and relatively low negative predictive value suggest that the absence of a detectable abnormality on TDI does not reliably exclude myocardial involvement. These estimates should also be interpreted cautiously because of the small sample size.
An important finding was the additional detection of myocardial abnormalities by TDI compared with conventional visual assessment. Of the 17 patients with RWMAs detected by pulsed systolic TDI, 9 (53%) did not have an identifiable wall motion abnormality on visual assessment of 2D echocardiography, whereas 8 (47%) demonstrated abnormalities by both methods. Thus, within the subgroup with TDI-detected abnormalities, more than half had no corresponding visually apparent RWMA on conventional 2D echocardiography. This finding suggests that quantitative assessment of myocardial systolic velocity may identify subtle regional dysfunction that is not readily apparent on routine visual wall motion analysis. TDI may therefore have a complementary role when conventional echocardiographic findings are inconclusive or visually normal despite biochemical evidence of myocardial injury.

Despite the association between Troponin I concentration and the presence of TDI-detected RWMA, Pearson's correlation analysis did not demonstrate a significant linear correlation between troponin I levels and the quantified segmental RWMAs. Therefore, although higher troponin I categories were associated with a greater frequency of detectable RWMA, the magnitude of troponin elevation did not show a corresponding linear relationship with the degree of regional myocardial dysfunction measured by TDI. This distinction is important because troponin I reflects myocardial injury, whereas tissue Doppler systolic velocity reflects regional myocardial mechanical function; these measures therefore represent different aspects of the pathophysiological consequences of acute myocardial ischemia.[11,12]

The absence of a significant linear correlation may also be influenced by several factors, including the timing of troponin measurement relative to symptom onset, infarct size and location, the number of myocardial segments involved, and the technical characteristics of TDI. In addition, the relatively small study population and wide distribution of troponin I values may have limited the ability to demonstrate a quantitative relationship between these variables. Consequently, larger studies using standardized imaging protocols and serial biomarker measurements are required to characterize this relationship more precisely.

Increasing evidence suggests that quantitative echocardiographic techniques may provide information complementary to conventional imaging and potentially contribute to clinical assessment and outcome prediction [3]. However, definitive systolic tissue velocity cut-off values for identifying CAD and regional myocardial dysfunction remain uncertain, and further studies are required to establish standardized values for the identification and quantification of RWMAs using TDI [5]. Abnormal tissue velocities may potentially reflect myocardial dysfunction beyond that evident from visual wall motion assessment alone, although this possibility cannot be established from the present data.[13,14]

Limitations:
Several limitations should be considered when interpreting the findings of this study. First, the sample size was small, with only 25 patients with NSTEMI. The resulting estimates of sensitivity, specificity, predictive values, and potential troponin I thresholds may therefore be imprecise and require validation in larger populations.

Second, tissue Doppler velocity measurements are angle dependent and principally measure myocardial motion along the ultrasound beam. This limitation can complicate assessment of multiple myocardial segments, particularly the more apical segments, and may affect the accuracy and reproducibility of regional myocardial velocity measurements.

Third, coronary angiography was not performed in all patients. Consequently, the location and severity of angiographically documented CAD could not be systematically compared with the distribution or magnitude of RWMAs quantified by TDI. Such a comparison would have strengthened assessment of the diagnostic relevance of the observed tissue velocity abnormalities.

Finally, 10 patients had systemic hypertension. Hypertension and associated left ventricular hypertrophy may themselves reduce tissue Doppler velocities and could therefore potentially confound the relationship between reduced systolic

myocardial velocities and ischemic myocardial dysfunction. Future studies should account for these potential confounding factors and include larger, well-characterized patient populations.

CONCLUSION

Pulsed systolic tissue Doppler imaging enabled quantitative identification of regional myocardial dysfunction in a substantial proportion of patients with NSTEMI and detected abnormalities in some patients without visually apparent RWMAs on conventional 2D echocardiography. Quantifiable RWMAs were present in 17 of 25 patients (68%), and troponin I levels were significantly higher in patients with TDI-detected RWMAs than in those without RWMAs ($P<0.001$). Although the presence of RWMA was associated with higher troponin I levels, no significant linear correlation was demonstrated between troponin I concentration and the quantified segmental abnormalities. The current data also do not provide sufficient evidence to establish a troponin I value of 0.5 as a definitive threshold for TDI-detectable RWMA. Larger prospective studies incorporating standardized TDI measurements, conventional echocardiography, serial troponin assessment, and coronary angiographic findings are needed to define the diagnostic role and clinically relevant cut-off values of pulsed systolic TDI in NSTEMI.

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