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Echocardiographic comparison of regional wall motion abnormality between patients with acute anteroseptal and acute extensive anteior ST segment elevation myocardial infarction

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Abstract

Post myocardial infarction (MI) short and long term clinical outcome is largely determined by the size of the infarcted area. It is generally assumed that as the lead involvement in electrocardiography (ECG) is less in anteroseptal ST segment elevation myocardial infarction (AS-STEMI), where ST segment elevation (STE) is limited to leads V_1 to V_3, myocardial damage is likely to be less; and in extensive anterior STEMI (EA-STEMI), as the STE extends further upto V6, the myocardial damage is likely to be more. This study was intended to compare regional wall motion abnormality (RWMA) between acute anteroseptal STEMI and acute extensive anterior STEMI patients. 90 patients with AS-STEMI and 106 patients with EA-STEMI, admitted in between October 2012 and September 2013, were included. For each patient, a transthoracic echocardiogram (TTE) was performed within 24-48 hours of MI and was interpreted by an independent investigator blinded to the patient's ECG data. No differences were observed between the two groups in baseline characteristics; except AS-STEMI group had more patients with diabetes and EA-STEMI group had more patients with family history of coronary artery disease. Distribution, extent of wall motion abnormalities and mean number of total involved segments were similar between patients with AS-STEMI and those with EA-STEMI ($p>0.05$). Regarding regional dysfunction, the apical septal (99.1% vs. 92.2%, $p<0.05$) and apical (76.4% vs. 60.0%, $p<0.05$) segments were the only two segments that were affected significantly more in patients with EA-STEMI than in patients with AS-STEMI. So, the term AS-STEMI may be a misnomer, as it implies that only the anteroseptal segments of the left ventricle are involved. This study shows that regional dysfunction in patients with AS-STEMI extends beyond the anteroseptal region. So, any patients with anterior wall involvement, either anteroseptal or extensive anterior STEMI, should be treated with equal importance.

Introduction

Regional wall motion abnormality is one of the earliest features of acute MI even before the infarctive change is evident in standard ECG or by rise of cardiac biomarker. Echocardiography can detect RWMA and it is widely available, noninvasive and relatively cheap as well.[1] STEMI is the most lethal form of acute MI (AMI).[1]

Anterior ST segment elevation myocardial infarction (A-STEMI) involves the territory of the myocardium supplied by the major artery of the heart i.e. left anterior descending artery (LAD). It can be commonly classified as anteroseptal STEMI and extensive anterior STEMI.[2-4]

Conventionally, ECG leads are said to be oriented according to the anatomic zones of the left ventricle i.e. V_1–V_3 for anteroseptal zone, V_4–V_6 for apical or lateral zone, I and aVL for high lateral zone. However, these conventional electrodes cannot be pinpointed or placed directly upon the heart itself and are situated some distance away; therefore a large area of myocardial injury may be substantially attenuated on surface ECG.[3]

Shalev et al.[5] suggested that, only the anterior-septum is never exclusively affected in patients with AS-STEMI. Porter et al.[6] showed that STE in lead V_1 and lead V_2 is associated with different areas of infarction rather than septum. Bogaty et al.[7] suggested that AS-STEMI is associated with an area of infarction predominantly involving the apex. But the apex may be involved either in anteroseptal, extensive anterior, or left dominant system occlusion related inferolateral MI. Bandeali et al.[4] showed that AS-STEMI causes similar area of left ventricular involvement as that in EA-

STEMI. Aldrich et al.[8] suggested that the number of leads with ST segment elevation correlates with the infarct size in patients with A-STEMI, implying that AS-STEMI is associated with an infarct size smaller than that in patients with EA-STEMI. However neither angiographic nor echocardiographic data has correlated with either injury pattern with the expected location of injury.[5-7,9] Two explanations regarding this matter opposing each other have been given; i.e. AS-STEMI is associated with a relatively small area of infarction, or it may be a manifestation of a large area of infarction, caused by a proximal occlusion of a wrapping left anterior descending artery (LAD) where STE in inferolateral leads (leads V_4 to V_6) is cancelled by an opposing injury vector involving the basal segments, i.e. the anterosuperior leads (leads V_1 and V_2).[5,9-11]

If MI was confined to the anterior septum, as it is thought, in so called AS-STEMI, it would require an isolated occlusion of the principal septal artery, and such an occurrence is unusual. An infarction of the anterior septum also would be expected to implicate occlusion of proximal LAD and should therefore be extensive.[7] Zhong et al.[10] found that STE in lead V_1 occurs when LAD is occluded proximal to first septal branch (S_1), which indicates that large area of myocardium is jeopardized which is perfused distal to S_1 and moreover, no significant difference was observed regarding the occlusion site between AS-STEMI and EA-STEMI in relation to the S_1 or first diagonal branch (D_1) of LAD.

Whether AS-STEMI is truly an infarction involving smaller area of the left ventricular myocardium or it is as extensive as EA-STEMI has not been well studied. The few studies carried out abroad were done with small sample size i.e. less than 100 patients. This study was intended to compare echocardiographic RWMA in acute AS-STEMI and acute EA-STEMI patients with larger sample size.

Materials and Methods

This cross sectional analytical study was carried out in the Department of Cardiology, Dhaka Medical College Hospital, over a period of one year, from October, 2012 to September, 2013. By purposive sampling technique, 196 patients were selected. Anterior STEMI was defined as diagnostic new STE at the J point in at least 2 contiguous leads of 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V_2–V_3 and/or of 1 mm (0.1 mV) in other contiguous chest leads or the limb leads, provided there was no evidence of left ventricular hypertrophy or left bundle-branch block.[12] AS-STEMI was denoted if STEMI was confined to leads V_1-V_3 and EA-STEMI was diagnosed when STEMI was confined to leads V_1-V_6,±I, aVL.[2,3,12]

Patients with history of old myocardial infarction/ intracoronary intervention/ coronary artery bypass grafting, STE in ECG other than MI i.e. pericarditis, prinzmetal angina, Brugada syndrome etc., ECG evidence of LBBB, WPW syndrome, ventricular arrhythmia, advanced second degree or third degree conduction defect, ventricular electronic pacing of heart, patients with cardiomyopathy, valvular heart disease, severe co-morbid conditions such as ESRD, cirrhosis of liver, malignancy etc., patients who were not thrombolysed, and patients with poor echocardiographic windows were all excluded.

ECG was done immediately after admission and was interpreted by an expert cardiologist. TTE was done between 24-48 hours of MI by an expert echocardiographer blinded to the ECG diagnosis. These patients were categorized into two groups. Those with STE confined to leads V_1-V_3 were denoted as group I (anteroseptal STEMI or AS-STEMI) and those with STE in leads V_1-V_6±I, aVL were denoted as group II (extensive anterior or EA-STEMI).

The left ventricle was divided into 17 segments (six basal, six mid-ventricular, five apical).[13] RWMA by TTE was assessed from each study subject according to the 17 segment model of left ventricle (Fig.1) in parasternal long axis, parasternal short axis, apical two chamber and apical four chamber views. RWMA detected in at least one segment was considered as involved and the results were recorded as normal, hypokinetic, akinetic and dyskinetic wall motion. Comparison of RWMA between the two groups for each of the 17 segments was done. Global wall motion abnormality was compared between the groups on the basis of ejection fraction (EF%).

Data were expressed as mean ± SD for continuous variables and as numbers (percent) for categorical variables. Continuous variables were compared by the paired-samples student t-test. Proportions were compared by Chi-square statistics; Fisher's exact test was used where appropriate. The 95% confidence intervals (CIs) were calculated for each technique. Differences were considered significant at the 0.05 level and the power of the study was set as 90%. All statistical calculations were performed using SPSS for Windows (version 16).

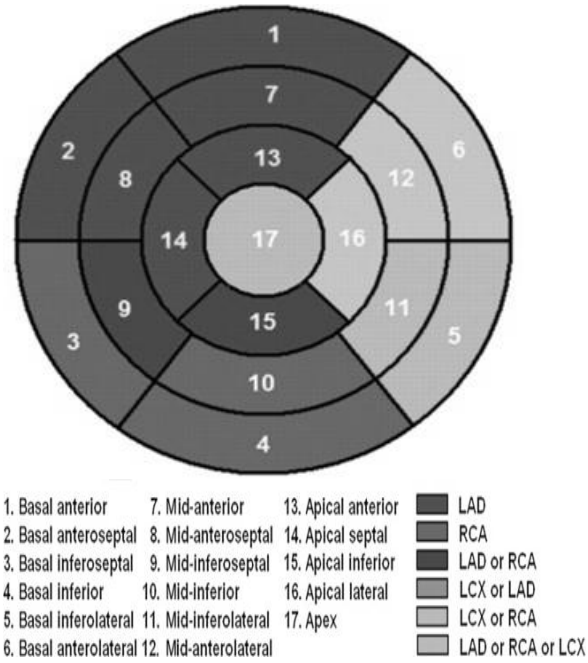

Fig.1: 17 segment model of left ventricle with respective coronary circulation.[13]

Results

The age distribution of the study subjects was similar in both AS-STEMI and EA-STEMI groups [mean (±SD) age 52.58 (±12.02) years vs. 50.72 (±13.22) years, p>0.05]. Gender distribution was similar in both AS-STEMI and EA-STEMI groups (91.1% male and 8.9% female vs. 83% male and 17% female, p>0.05).

In group I, 54(60.0%) patients and in group II, 36 (34.0%) patients had history of diabetes mellitus. Group I had significantly more diabetic patients (60% vs. 34%, p<0.05). Group II had significantly more patients with family history of CAD (31.1% vs. 47.2%, p<0.05). No statistically significant difference was observed regarding obesity, hypertension, dyslipidaemia, sedentary life style, and smoking status between two groups.

More than 90% of the study subjects in both groups had predominant involvement of mid anterior, mid anteroseptal, mid inferoseptal, apical anterior, apical septal walls, and a significant number of study subjects had basal anterior, basal anteroseptal, apical anterior, apical septal segments involvement, indicating typical involvement of LAD territory. Variable degree of regional wall motion abnormality is seen in other segments indicating involvement of overlapping zones of LAD-RCA (right coronary artery) and LAD-LCX (left circumflex) artery supply.

In the present study, mean (±SD) number of total involved segments in group I and group II patients were 8.83(±2.49) and 9.01(±2.25) respectively, with no statistically significant difference (p>0.05) (Table-I). When research assessed regional dysfunction, the apical septal or segment 14 (99.1% vs. 92.2%, p<0.05) and apical or segment 17(76.4% vs. 60.0%, p<0.05) were the only two segments that were affected significantly more in patients with EA-STEMI than in patients with AS-STEMI (Table-II).

There was no significant difference in global wall motion abnormality as assessed by measuring mean (±SD) ejection fraction (EF%) between patients with AS-STEMI or EA-STEMI [38.80 % (±5.78) and 39.21 % (±5.90); p>0.05] (Table-III).

Table I: Comparison of mean of total involved segments between two groups (n=196).

Total involved segments	Groups		P (95% CI)
	Group I (n=90)	Group II (n=106)	
Mean ±SD	8.83±2.49	9.01±2.25	0.604^{NS} (.492 to .498)
Range	3-15	4-15	

NS = not significant.

Table II: Comparison of presence of left ventricular segmental wall motion abnormality (hypokinetic, akinetic and dyskinetic wall motion) with respective echocardiographic left ventricular segments between two groups (n=196).

LV segmental wall motion abnormality		Groups		p
		Group I (n=90) n (%)	Group II (n=106) n (%)	
Segment 1	Basal anterior	38(42.2%)	35(33.0%)	0.184^{NS}
Segment 2	Basal anteroseptal	42(46.7%)	43(40.6%)	0.390^{NS}
Segment 3	Basal inferoseptal	34(37.8%)	48(45.3%)	0.288^{NS}
Segment 4	Basal inferior	8(8.9%)	6(5.7%)	0.382^{NS}
Segment 5	Basal inferolateral	2(2.2%)	2(1.9%)	0.869^{NS}
Segment 6	Basal anterolateral	4(4.4%)	1(0.9%)	0.121^{NS}
Segment 7	Mid- anterior	86(95.6%)	100(94.3%)	0.700^{NS}
Segment 8	Mid -anteroseptal	88(97.8%)	104(98.1%)	0.869^{NS}
Segment 9	Mid -inferoseptal	88(97.8%)	100(94.3%)	0.225^{NS}
Segment 10	Mid- inferior	16(17.8%)	20(18.9%)	0.844^{NS}
Segment 11	Mid- inferolateral	2(2.2%)	6(5.7%)	0.225^{NS}
Segment 12	Mid-anterolateral	56(62.2%)	68(64.2%)	0.780^{NS}
Segment 13	Apical anterior	88(97.8%)	104(98.1%)	0.869^{NS}
Segment 14	Apical septal	83(92.2%)	105(99.1%)	0.016^{S}
Segment 15	Apical inferior	34(37.8%)	40(37.7%)	0.995^{NS}
Segment 16	Apical lateral	72(80.0%)	92(86.8%)	0.200^{NS}
Segment 17	Apical	54(60.0%)	81(76.4%)	0.013^{S}

NS = not significant, S = significant.

Table III: Comparison of mean of ejection fraction between two groups (n=196)

Ejection fraction	Groups		P (95% CI)
	Group I (n=90)	Group II (n=106)	
Mean ± SD	38.8±5.78	39.21±5.90	0.627^{NS} (-2.059 to 1.245)

NS = not significant, S = significant

Discussion

Age distribution in both groups in our study was similar to Bandeali et al.[4], Porter et al.[6], Huang et al.[9], Zafrir et al.[14] and similarity in gender distribution was also found between our study and several other similar studies.[4,6,9]

Comparison of traditional risk factors revealed that AS-STEMI group had significantly more patients with diabetes mellitus and family history of CAD was significantly more prevalent in EA-STEMI patients. But, as a whole, no significant difference was observed regarding the prevalence of smoking, hypertension, dyslipidaemia, sedentary life style and obesity between the groups, as was observed by Huang et al.[9]

The results of our study showed that there was no significant difference in global wall motion abnormality as assessed by measuring ejection fraction between patients with AS-STEMI and EA-STEMI. Our finding is consistent with Bandeali et al.[4] who reported mean (±SD) EF% was 39.17% (±10.5) vs. 44.08% (±10.5) with p>0.05. The same was found by some other researchers.[6]

In the present study, mean (± SD) number of total involved segments in group I and group II patients were 8.83 (±2.49) and 9.01 (±2.25) respectively with no statistically significant difference. This finding was supported by Bandeali et al.[4] where mean number of total involved segments were 6.5 (±4) and 8 (±4.5) in AS-STEMI and EA-STEMI groups respectively with no statistically significant difference.

When we assessed regional dysfunction, the apical septal or segment 14 and true apical or segment 17 were the only two segments that were affected significantly more in patients with EA-STEMI than in patients with AS-STEMI. In patients with EA-STEMI, a trend was observed toward a greater degree of regional dysfunction in the basal inferoseptal or segment 3(45.3% vs. 37.8%, p>0.05), mid inferolateral or segment 11(5.7% vs. 2.2%, p>0.05), mid anterolateral or segment 12 (64.2% vs. 62.2%, p>0.05), apical lateral or segment 16(86.8% vs. 80.0%, p>0.05) but the differences were not statistically significant. On the other hand, in patients with AS-STEMI, a trend was observed toward a greater degree of regional dysfunction in the basal anterior or segment 1 (42.2% vs 33.0%, p>0.05), basal anteroseptal or segment 2(46.7% vs 40.6%, p>0.05), basal inferior or segment 4(8.9% vs 5.7%, p>0.05), basal anterolateral or segment 6(4.4% vs 0.9%, p>0.05), mid inferoseptal or segment 9 (97.8% vs 94.3%, p>0.05), but the differences were not statistically significant. The incidence of the involvement of basal inferolateral or segment 5(1.9% vs 2.2%, p>0.05), mid anterior or segment 7(94.3% vs 95.6%, p>0.05), mid anteroseptal or segment 8 (98.1% vs 97.8%, p>0.05), mid inferior or segment 10(18.9% vs 17.8%, p>0.05), apical anterior or segment 13(98.1% vs 97.8%, p>0.05), apical inferior or segment 15(37.7% vs 37.8%, p>0.05) were similar between patients with EA-STEMI and AS-STEMI.

More than 90% of the study subjects in both groups had predominant involvement of mid anterior, mid anteroseptal, mid inferoseptal, apical anterior, apical septal walls, and a significant number of study subjects had basal anterior, basal anteroseptal, apical anterior, apical septal segments involvement, indicating typical involvement of LAD territory. Variable degree of regional wall motion abnormality seen in other segments indicates involvement of overlapping zones of LAD-RCA and LAD-LCX artery supply.

Our study was in agreement with Bandeali et al.[4] They studied 65 patients with anterior STEMI who underwent TTE within 24-48 hours of admission. They also showed a significant proportion of the subjects having involvement of mid anterior, mid anteroseptal, mid inferoseptal, apical anterior, apical septal, true apical segments and a moderate number of study subjects having involvement of basal anterior, basal anteroseptal segments. This finding was consistent with the current study with the exception of apical inferior segment (segment 15) where dysfunction occurred more often in patients with EA-STEMI than in patients with AS-STEMI (71.4% vs. 43.3%; p<0.05). The discrepancy can be explained by the presence of an alternative blood supply to the apical segments (because of the presence large diagonal or obtuse marginal branches or even branch of RCA), apical areas may be spared or involved in varying degree; moreover apex is an overlapping zone of LAD-RCA and LAD-LCX arteries.[15]

Huang et al.[9] reported two contrasting angiographic findings and their relation to left ventricular segments of involvement among patients with A-STEMI. First, many patients with AS-STEMI than patients with EA-STEMI (52% vs. 15%) had proximal occlusion of a short LAD which did not reach the apex and an alternative blood supply to the apex by a large side branch, indicating more involvement of basal and mid segments sparing apical segments. In contrast, more patients with AS-STEMI than patients with EA-STEMI (52% vs. 15%) had a proximal occlusion of a long wrap-around LAD, suggesting extensive left ventricular area of involvement. This is consistent with the current study because of the similarities in wide

range of involvement of left ventricular segments, variable involvement of apical segments. Their angiographic findings are supported by the echocardiographic findings of our study, suggesting AS-STEMI is not always limited to the anteroseptal segment.

Moreover, Porter et al.[6] showed comparable global left ventricular function and regional dysfunction irrespective of the presence of STE in lead V_5 or V_6. No difference in regional dysfunction was observed between the patients with AS-STEMI and EA-STEMI showing similar involvement of basal anterior, basal anteroseptal, basal inferoseptal, and apical inferior segments between the groups without any statistical significance. Involvement of basal segments indicates proximal occlusion of LAD and extensive damage to left ventricle in both AS-STEMI and EA-STEMI. All these evidences make it clear that each electrode records the global activation vector toward and away from each lead and not the local events adjacent to the electrode. Therefore, leads V_1 to V_3 do not represent only the anteroseptal regions. Therefore more sophisticated classification of anterior STEMI had been suggested.[14]

Zafrir et al.[14] performed 17 segment based myocardial perfusion scan to see left ventricular segmental involvement in 55 patients with A-STEMI. The study showed involvement of basal segments occurring predominantly in AS-STEMI patients. On the contrary, mid and apical segmental dysfunction was more in EA-STEMI. This finding suggested more proximal occlusion of LAD in AS-STEMI than in EA-STEMI. No relation was observed regarding more lateral involvement with the presence of STE in leads I, aVL.

Engelen et al.[11] also studied 100 patients of acute anterior STEMI to find out the difference of occlusion site in AS-STEMI and EA-STEMI and found no difference in occlusion site between the groups. This finding also implied that AS-STEMI is not a MI involving areas less than EA-STEMI.

Conclusion: The present study concludes that RWMA and global LV dysfunction in ECG pattern of acute AS-STEMI is not less extensive than that in the patients with the ECG pattern of acute EA-STEMI. The term AS-STEMI neither implies that the ischemic process is limited to the anteroseptal segments, nor that the size of the ischemic area at risk is smaller than that in patients with EA-STEMI and it is as extensive as EA-STEMI. Both are equally devastating for patients. Therefore, the terminology "anteroseptal" is probably a misnomer and should not downgrade the extent of myocardial involvement as compared to that in EA-STEMI. So, any patients with anterior wall involvement, either AS-STEMI or EA-STEMI, should be treated with equal importance.

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