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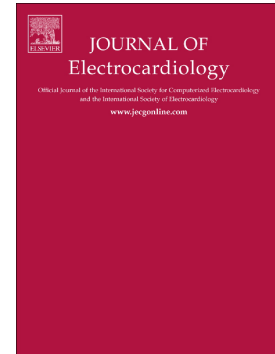
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**Correlation of anteroseptal ST elevation with myocardial infarction territories  
through cardiovascular magnetic resonance imaging**

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Running head – Addressing nomenclature of anteroseptal STEMI through CMR  
correlations

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## ABSTRACT

### *Background*

Anteroseptal ST elevation myocardial infarction (STEMI) is traditionally defined on the electrocardiogram (ECG) by ST elevation (STE) in leads V1-V3, with or without involvement of lead V4. It is commonly taught that such infarcts affect the basal anteroseptal myocardial segment. While there are suggestions in the literature that Q waves limited to V1-V4 represent predominantly apical infarction, none have evaluated anteroseptal ST elevation territories. We compared the distribution of the myocardium at risk (MaR) in STEMI patients presenting with STE limited to V1-V4 and those with more extensive STE (V1-V6).

### *Methods*

We identified patients in the MITOCARE study presenting with a first acute STEMI and new STE in at least two contiguous anterior leads from V1 to V6. Patients underwent cardiac magnetic resonance (CMR) imaging three to five days after acute infarction.

### *Results*

Thirty-two patients met inclusion criteria. In patients with STE in V1-V4 (n=20), myocardium at risk (MaR) > 50% was seen in 0%, 85%, 75%, 100%, and 90% in the basal anteroseptal, mid anteroseptal, apical anterior, apical septal segments, and apex, respectively. The group with STE in V1-V6 (n=12), MaR > 50% was seen in 8%, 83%, 83%, 92%, and 83% of the same segments.

### *Conclusions*

Patients with acute STEMI and STE in leads V1-V4, exhibit MaR in predominantly apical territories and rarely in the basal anteroseptum. We found no evidence to support

existence of isolated basal anteroseptal or septal STEMI. “Anteroapical” infarction is a more precise description than “anteroseptal” infarction for acute STEMI patients exhibiting STE in V1-V4.

## 1. INTRODUCTION

ST-elevation (STE) isolated to leads V1 to V3 with or without involvement of lead V4 in the electrocardiogram (ECG) have traditionally defined “anteroseptal” ST elevation myocardial infarction (STEMI).[1] This definition was derived decades ago from histopathological studies, correlating the distribution of Q waves and the extent of infarction, and its validity has been questioned given the selection bias native to such works.[2, 3]

Later investigations have used cardiac imaging to correlate ECG infarct patterns with the purported infarct territories. The common belief in isolated anteroseptal myocardial infarction (MI) was not supported by early echocardiographic and angiographic data, and the inability of echocardiography to distinguish between stunned, hibernating and infarcted myocardium was a limiting factor in these investigations.[4, 5] Ultimately Selvanayagam et al. evaluated a group of 19 patients with acute MI and new Q waves in V1 to V2-V4 with LGE MRI, and reported that the majority evidenced mostly apical infarction with some mid-ventricular anteroseptum involvement.[6]

Cardiovascular magnetic resonance (CMR) allows for assessment of myocardium at risk (MaR) and MI. Myocardial edema is precipitated by acute coronary thrombosis, resulting in an increased myocardial extracellular volume fraction (ECV) in the affected region, referred to as MaR. The increased ECV within the MaR can be depicted by contrast-enhanced steady state free precession (CE-SSFP) up to one week after the acute event.[7] Myocardial ischemia precedes the development of infarction, which can be perceived through late gadolinium enhancement cardiac MRI (LGE MRI), an exquisitely



sensitive method for assessment of the size and extent of infarcted myocardium.[8] The MaR may thus be visualized and compared to the region of subsequent infarct.[7]

We aimed to evaluate the location and extent of MaR in patients fitting the conventional definition of “anteroseptal” STEMI and those with more extensive STE in the precordial leads. We hypothesized that the ECG term “anteroseptal MI” actually refers to an area of MaR that is mostly apical, rather than basal, as traditional pedagogy sets forth. To our knowledge, this is the first work to directly correlate ECG findings of anteroseptal STEMI and areas of MaR.

## 2. METHODS

Patients in our work were participants in the MITOCARE study, a phase II, multicenter, randomized, double-blind, placebo-controlled study assessing the safety and efficacy of TRO40303 for reduction of reperfusion injury in patients undergoing percutaneous coronary intervention (PCI) for acute STEMI. [9, 10]

### *Patient population*

Inclusion criteria for the original study were first acute STEMI with occlusion of either the left anterior descending artery (LAD), the right coronary artery (RCA) or the left circumflex (LCX) artery; nitrate resistant chest pain for at least 30 minutes; presentation within six hours of onset of chest pain; new ST elevation at J-point in two contiguous leads with cut-off points:  $>0.2$  mV in males or  $>0.15$  mV in females in leads V2-V3 and/or  $>0.1$  mV in other leads. Patients with cardiac arrest, cardiogenic shock, previous acute MI, previous coronary artery bypass graft, intravenous fibrinolytic therapy within 72 hours prior to PCI were excluded. Additional details of the MITOCARE study

have been previously published in full.[9, 10]. In the present study we included patients with anterior myocardial infarction presenting with ST elevation in two or more of the precordial leads V1-V3 with or without ST elevation in additional precordial leads. Patients with wide QRS or electronic paced rhythm on the presenting ECG were excluded.

Electrocardiograms were recorded at hospital admission, with analyses performed in the ECG core laboratory at Rigshospitalet (Copenhagen, Denmark). ST-segment deviation was measured manually to the nearest 0.5 mm at the J-point in all 12 ECG leads. Three to five days after the acute infarction, CMR was performed on whole-body 1.5 T magnetic resonance scanners with cardiac applications used for standard clinical CMR.

#### *Cardiac magnetic resonance imaging*

*Image acquisition:* The MR images were acquired in multiple CMR-centers (n=10) across Europe on either a 1.5T or 3T MR scanner. CE-SSFP short-axis cine images of the left ventricle were obtained five minutes after intravenous injection of a gadolinium-based contrast agent. Approximately 15 minutes after the contrast injection, LGE images were acquired in the same image planes as the CE-SSFP.[9, 11]

*Image analysis:* Left ventricular function and MaR were assessed from the CE-SSFP short-axis cine images. The endo- and epicardial borders as well as MaR were manually delineated in both end-systole and end-diastole as previously described.[7] MaR was expressed both as a percentage of the LV and as a percentage of each of the 17 segments pertaining to the American Heart Association (AHA) 17-segment model for

uniform description of myocardial segments.[12] Infarct size was assessed by LGE CMR.[13] CMR analyses were performed by a core laboratory (Imacor AB, Lund, Sweden: [www.imacor.se](http://www.imacor.se)) blinded to all clinical data using the software Segment ([www.medviso.com](http://www.medviso.com)).[14]

### *Statistical methods*

The Fisher's exact test was carried out to compare categorical variables. Continuous variables were compared with one-way analysis of variance (ANOVA). A  $p$ -value  $<0.05$  was considered to be statistically significant. When appropriate, values are described as mean  $\pm$  standard deviation. Frequencies and percentages have been used to describe categorical variables.

## **3. RESULTS**

### *Patient Characteristics*

A total of 32 patients met inclusion criteria.[9] The mean age of the patients was  $63 \pm 11$  years. Nineteen patients (59%) were male (Table 1).

### *Principal analysis*

Patients were categorized according to the distribution of anterior STE on the admission ECG. Twenty patients had STE extending up to V4 (two patients with STE in V1-V2, two with STE in V1-V3, 14 with STE in V1-V4, and two with STE in V2-V4). Twelve patients had STE extending up to V6 (five with STE in V1-V5, two with V1-V6, five with V2-V5).

Mean left ventricular ejection fraction (LVEF) assessed by CMR was  $46.0\% \pm 10.2\%$ .

There was no statistically significant difference in the LVEF between the two groups (Table 1)

Patients with extensive anterior STEMI (STE beyond V4) exhibited greater percentage of MaR ( $p = 0.04$ ) as well as mass of MaR ( $p = 0.0006$ ) (Table 1).

Figure 1 illustrates the extent of myocardial edema across the 17 myocardial segments in the two groups. The apical lateral myocardial segment (segment 16) was affected more frequently in patients with extensive anterior STEMI, but otherwise there were no statistically significant differences in the segmental involvement between the two groups.

In patients with anterior STE extending to V4 ( $n = 20$ ), MaR  $>25\%$  was seen in seven (35%), nineteen (95%), eighteen (90%), twenty (100%), and twenty (100%) patients in the basal anteroseptal, mid anteroseptal, apical anterior, apical septal segments, and apex, respectively (Figure 1a). MaR  $>50\%$  was seen in zero (0%), seventeen (85%), fifteen (75%), twenty (100%), and eighteen (90%) (Figure 1b) at the same segments.

In patients with extensive anterior STEMI ( $n = 12$ ), three (25%), ten (83%), eleven (92%), eleven (92%), and eleven (92%) patients were found to have MaR  $>25\%$  at the basal anteroseptal, mid anteroseptal, apical anterior, apical septal segments, and apex, respectively (Figure 1a). A similar pattern was observed when comparing the distribution of segments with  $>50\%$  MaR involvement: one (8%), ten (83%), ten (83%), eleven (92%), and ten (83%) patients, respectively (Figure 1b).

A separate analysis of these same study patients was carried out to examine whether STE in lead V1 is associated with involvement of the basal segments. All study patients

were regrouped into those with STE in V1 (n= 25) and those without STE in V1 (n = 7). There was no statistically significant difference in frequency of MaR >50% for any segments of the basal myocardium between these groups, with infrequent involvement noted overall (Table 2). Similar results were found for MaR >25% (data not shown).

#### 4. DISCUSSION

Conventional ECG nomenclature with regard to STEMI diagnosis has been based on old histopathological correlations, which are inherently skewed due to the inherent selection bias of such works. Advanced CMR techniques permit the quantitative and qualitative assessment of MaR and infarction with higher sensitivity than other modalities.[15]

To our knowledge, our work is the first to directly examine correlations between MaR and ECG distributions of STE in the various leads. Selvanayagam et al., using LGE MRI, demonstrated principally apical infarction and some involvement of the mid-ventricular anteroseptum in 19 patients with Q-wave MI restricted to leads V1 to V2-V4.[6] The basal anteroseptum was not affected. Patients with Q waves limited to V1-V3 (n=8) and more extensive anterior Q waves (n=11) were not separately analyzed. Our findings with the presenting ECG are in general agreement with those of Selvanayagam et al in the chronic phase of infarction, suggesting that the pattern called “anteroseptal” STEMI is actually anteroapical STEMI.

To categorize the ECGs of the study patients, we used the classical definition of “anteroseptal” infarction, namely, STE in leads V1 to V4.[16-18] Figures 2 and 3 depicts the CMR images and ECGs of patients with limited and extensive anterior STE.

A large advantage of our study is that we were able to include patients with more extensive anterior STE, from leads V1 to V6 and compare to those with more limited “anteroseptal” STE, which has not been done in any prior work. The distributions of myocardial edema (Figure 1) suggest that more extensive anterior STE are correlated with overall increased MaR. However, it should be noted that the apical segments are affected in both “anteroseptal” and extensive anterior STEMI and the only statistically significant difference is more apical lateral segment involvement in the V1-V6 group. A minority of patients in both groups evidenced MaR in the basal anteroseptal segments. There was a dominant involvement of the apical anterior (segment 13), septal (segment 14), and apex (segment 17) in both groups, and the mid anterior (segment 7) mid ventricular anteroseptum (segment 8) were also commonly affected.

Quite strikingly, no patient in this study exhibited isolated basal anteroseptal (segment 2) MaR, in contrast to the prevailing theories in the literature. An example of this is seen in a prominent American Heart Association (AHA) ECG interpretation course, which instructs learners that “anteroseptal infarction” is limited to the basal anteroseptum.[19]

The left anterior descending (LAD) artery branches from the left main coronary artery (LM), coursing along the anterior interventricular groove toward the apex, supplying branches to the septum and the free wall of the left ventricle (LV).[16] Occlusion of the LAD would put the apical segments at risk, except in cases where the LAD is short, and there is an alternate blood supply to the apex. Because apical involvement is to be expected in LAD occlusion, the significance of STE in precordial leads beyond V1-V4 is unclear. Huang et al. suggested that the lack of STE in leads V4-

V6 in some patients with “anteroseptal” STEMI may actually signify cancellation of the opposing apical and basal-anterior injury vectors (caused by proximal occlusion of a long LAD), rather than a smaller MaR.[20] Yet, our study showed basal involvement in only a small proportion of patients (Figure 1).

A granular categorization of ECG anterior infarction patterns has been suggested by others, such as “septal,” “apical anteroseptal,” and “limited anterior.”[21, 22] Our data demonstrate that the myocardium appears to be principally at risk at the apical segments. Thus, we believe that “anteroapical” infarction should replace the term “anteroseptal” infarction, supporting the propositions made by Shalev et al. and Bogaty et al in the more chronic stage of infarction.[4, 5]

Ben-Gal et al. suggested that STE in lead V1 is precipitated by ischemia of the basal septum as a result of involvement of the proximal septal branches of the LAD in patients without a large conal branch of the RCA.[23, 24] The group suggested that the absence of STE in lead V1 implies the presence of a large conal branch of the RCA supplying the basal part of the interventricular septum. We in fact found minimal involvement of the basal segments with no significant differences between segments on separate analysis of patients with and without STE in V1 (Table 2).

#### *Future directions*

Further investigations in traditional ECG terminology are needed, especially territorial definitions of MI. CMR allows for noninvasive assessment of several infarction parameters without need for radiation and is an exquisitely useful tool in this regard. Larger studies will be of great value. While the expense of CMR poses a barrier to

adoption, wider availability, especially in larger medical centers, may facilitate its use in appropriate clinical scenarios.

#### *Study limitations*

Due to the small number of patients with limited “septal” STE in V1-2, we were unable to do further subgroup data analysis. Further investigations in this domain are warranted, as the field of CMR remains fertile.[15] Also, anteroseptal STE have been reported in conus branch artery occlusion.[25]

#### *Conclusion*

Acute STEMI with STE in leads V1-V3/4 primarily affects the apical myocardial territories (apical septal, apical anterior, and apex). Rarely is the basal interventricular septum involved. This work refutes the existence of lone basal anteroseptal or septal MI. Thus “anteroapical” is a more descriptive label than “anteroseptal” for an ECG with STE in V1-V3/V4 in a patient with acute MI.

#### *Acknowledgements*

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#### *Conflicts of Interest*

Einar Heiberg is founder of Medviso AB, developing cardiovascular image processing software. Marcus Carlsson and Henrik Engblom are consultants at Imacor AB. Håkan Arheden is founder of and employed by Imacor AB.



## TABLES AND FIGURES

**Table 1.** Patient characteristics in the anterior ST-elevation (STE) distributions. Where applicable, values are reported as mean  $\pm$  standard deviation. *P* values were produced by analysis of variance (ANOVA) comparing sample means among the three groups. IS = infarct size; MaR = myocardium at risk.

	ECG STE distribution		<i>P</i> value	All groups
	V1-V4	V1-V5/V6		
<b>Number of patients</b>	20	12	-	32
<b>Age (years)</b>	64.7 $\pm$ 11.2	60.3 $\pm$ 11.1	0.42	63.1 $\pm$ 11.0
<b>Male, n (%)</b>	12 (60)	7 (58)	0.61	19 (59)
<b>Tobacco use history, n (%)</b>	10 (50)	7 (58)	0.46	17 (53)
<b>Myocardial segments with edema (n)</b>	10.1 $\pm$ 1.71	11 $\pm$ 1.8	0.26	10 $\pm$ 1.8
<b>Myocardium at risk (%)</b>	38.7 $\pm$ 9.49	45.6 $\pm$ 8.08	0.04	41.3 $\pm$ 9.48
<b>Myocardium at risk (g)</b>	47.4 $\pm$ 11.8	64.8 $\pm$ 13.5	0.0006	53.9 $\pm$ 14.9
<b>IS/MaR (%)</b>	43.8 $\pm$ 20.0	54.0 $\pm$ 13.7	0.15	47.6 $\pm$ 18.4
<b>LVEF (%)</b>	47.7 $\pm$ 9.82	42.3 $\pm$ 10.2	0.16	46.0 $\pm$ 10.2

**Table 2.** A comparison patients with ST elevation in V1 and those without. Frequency of myocardium at risk >50% in the basal myocardium for study patients is described. *P* values were computed by Fisher's exact test.

Segment number	Myocardial Segment	ECG STE distribution		<i>P</i> value
		STE in V1, n (%)	No STE in V1 n (%)	
1	Basal anterior	0 (0)	0 (0)	1
2	Basal anteroseptal	2 (8)	0 (0)	0.61
3	Basal inferoseptal	0 (0)	0 (0)	1
4	Basal inferior	0 (0)	0 (0)	1
5	Basal inferolateral	0 (0)	0 (0)	1
6	Basal anterolateral	0 (0)	0 (0)	1

**Figure 1.** An illustration of myocardial segments evidencing >25% myocardium at risk (A) and >50% myocardium at risk (B) assessed by cardiac magnetic resonance (CMR) imaging across the entire study group. Patients are grouped by ECG ST elevation (STE) distribution (V1-V4 and V1-V5/6). *P* values were computed by Fisher's exact test. There was a significant difference in the extent of MaR at the apical lateral segment (\*) for MaR >25% ( $p = 0.017$ ) and MaR >50% ( $p = 0.049$ ). Other comparisons between segments were statistically nonsignificant.

**Figure 2.** Myocardium at risk (MaR) shown as contrast-enhanced area at MR in basal, mid-ventricular and apical short-axis views of a patient with STE in V1-V5 (a) and in a patient with STE limited to V1-V2 (b). Arrows show the MaR. Patient A has a larger MaR compared to patient B. Both patients have apical involvement; however, patient A (with STE in V1-V5) has additional involvement of the mid-anterior and mid-inferoseptal segments. Neither patient demonstrates any MaR in the basal anteroseptal or basal anterior wall.

**Figure 3.** Electrocardiograms of two study patients: one with extensive anterior ST elevations (V1-V6) (a) and the other with more limited anterior ST elevations (V1-V4) (b).

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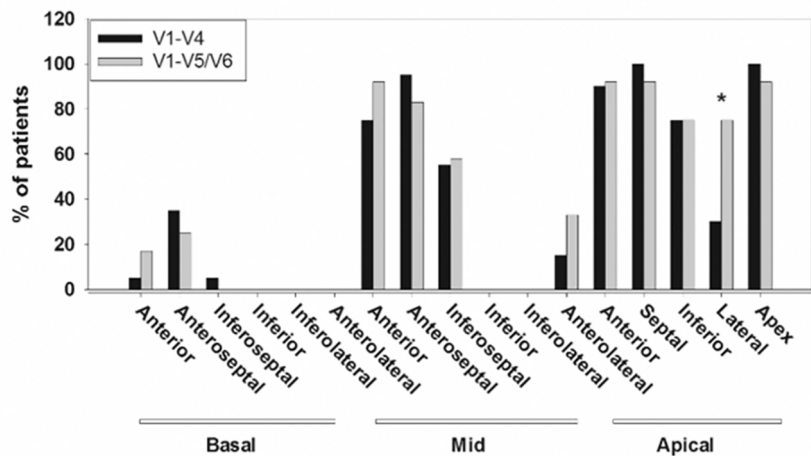
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### Highlights

- Acute MI with STE in V1-V4 exhibits MaR in predominantly apical territories
- Our data do not support existence of isolated basal anteroseptal or septal STEMI
- “Anteroapical” infarction is a more precise description than anteroseptal infarct



A



B

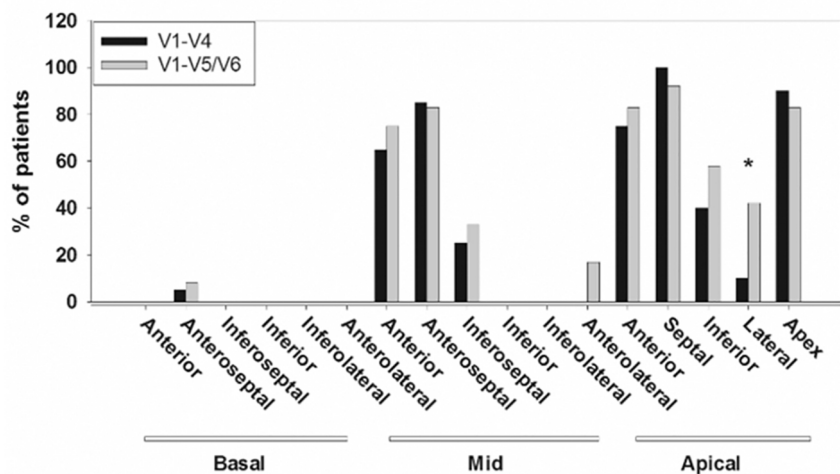
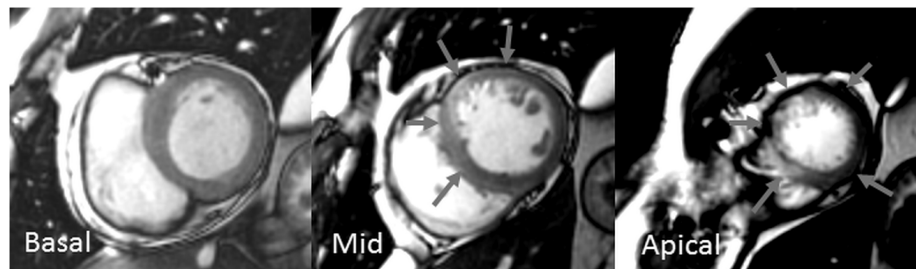
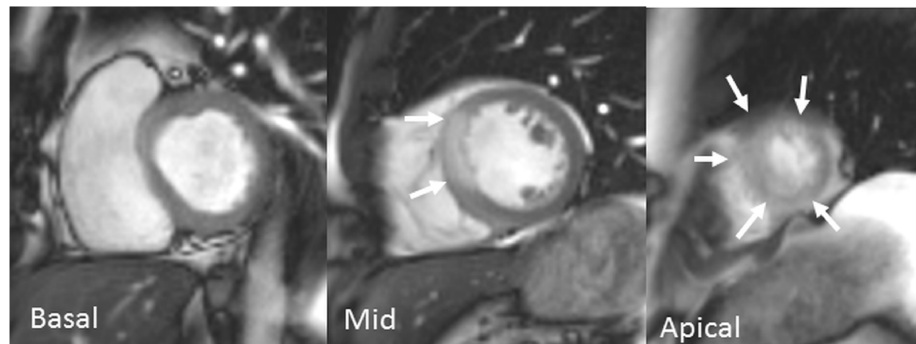


Figure 1



A



B

Figure 2

**a.**



**b.**



Figure 3