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VIABILITY

Anteroseptal or Apical Myocardial Infarction: A Controversy Addressed Using Delayed Enhancement Cardiovascular Magnetic Resonance Imaging[#]

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ABSTRACT

Aim: Delayed enhancement MRI (DE-MRI) of the heart has been shown to reliably identify areas of irreversible myocardial damage. We sought to determine if the term anteroseptal MI is appropriate by correlating electrocardiographic, angiographic, cine MRI and DE-MRI findings. *Methods and Results:* Nineteen patients admitted to our hospital with their first acute anterior MI and whose ECG showed new Q waves in leads V_1-V_4 were studied. All patients underwent cardiac catheterization, cine MRI, and DE-MRI. The mean left ventricular ejection fraction was 53%±16%. All 19 patients had evidence of delayed hyperenhancement in one or more myocardial segments (mean number of affected segments 5.5±2.1). The mean mass of hyperenhanced myocardium was 14±8 grams, or 10%±6% of absolute LV mass. Nineteen (100%) and 15 (79%) patients showed evidence of delayed hyperenhancement of the apex and apical anterior segments respectively. Seven (37%) patients showed evidence of mid ventricular anteroseptal hyperenhancement and none had any hyperenhancement of basal anteroseptal segments. *Conclusion:* High resolution cardiac MRI applied in patients with acute infarction and new Q waves in leads V_1-V_4 demonstrates the presence of predominantly apical, but not isolated septal or anteroseptal infarction.

Key Words: Electrocardiography; Magnetic resonance imaging; Acute myocardial infarction.

[#]No conflict of interest exists for any of the authors in relation to this study.

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INTRODUCTION

The use of the term anteroseptal infarction in instances where isolated Q waves are found in leads V_1-V_4 dates back to two studies performed more than 50 years ago: Wilson and Rosenbaum, proposed that the involvement of the precordial leads V_1-V_4 implied infarction of the anteroseptal left ventricular wall (Wilson and Rosenbaum, 1943). This assertion was confirmed by Myers and Stofer in a histopathologic study (Myers and Stofer, 1948). However, the selection bias in pathological studies and the different clinical features detailed in the case histories had raised questions as to the applicability of these findings to current clinical practice.

Shalev and colleagues compared the ECG, echocardiographic and cardiac catheterization findings of 80 patients who fitted the traditional definition of anteroseptal infarction and found that wall motion abnormalities were most frequently confined to the anteroapical region and that the septum was spared in >80% of patients (Shalev et al., 1995). In contrast, a more recent report by Bogarty et al., on 50 patients presenting with Q waves in V_1 to V_{2-4}, while again finding that the principal segment affected was the apex, found that the septum was involved in as many as 50% of patients (Bogaty et al., 2002).

A drawback of all the previous studies that have attempted to localize the site of infarction in such patients, are the inherent limitations of the imaging modality used to define infarcted myocardial territory. Echocardiographic image acquisition depends on the operator and the acoustic window (Grothues et al., 2002). In addition, wall motion abnormalities detected by echocardiogram and/or X-ray contrast ventriculography can reflect either stunned/hibernating (i.e., potentially viable) as well as irreversibly injured myocardium (Rahimtoola, 1989; Wijns et al., 1998). Furthermore, patients with isolated subendocardial myocardial infarction may well have this missed by imaging modalities such as single photon emission computed tomography (SPECT) as a result of its poor spatial resolution (Wagner et al., 2003).

Cardiovascular Magnetic Resonance Imaging (MRI) permits noninvasive assessment of cardiac function and viability with high spatial resolution (Fieno et al., 2000; Klein et al., 2002; Mahrholdt et al., 2002; Wu et al., 2001). In contrast to echocardiography and radionuclide studies, delayed enhancement MRI (DE-MRI) allows quantification of infarct tissue as well as assessment of the transmural extent of infarction (Wagner et al., 2003). Recent reports have confirmed the usefulness of DE-MRI in predicting recovery of regional wall function post revascularization and in localizing areas of infarction both in cases due to coronary disease and iatrogenic manipulations such as alcohol ablation (Kim et al., 2000; Sievers et al., 2002; Wu et al., 2001). Using this new high resolution technique for detection of myocardial viability, we revisited the true nature of anteroseptal infarction. Our hypothesis was that the classic definition of ''anteroseptal infarction'' is a misnomer and that anteroseptal infarction represents mainly apical rather than isolated septal or anteroseptal infarction.

METHODS

Patients

Thirty-eight consecutive patients admitted to our hospital with their first acute anterior MI and whose ECG showed by the third day, Q waves as defined by Minnesota code 1-1-1 to 1-2-7 in leads V_1-V_4 (Crow et al., 1997), were considered for possible inclusion in the study. Out of these 38 screened patients, 19 (50%) were included in the study population. We excluded the following patients: coronary angiogram not performed (14 patients), QRS duration >0.12 seconds (two patients), typical MRI contraindications (e.g., pacemaker, implantable defibrillator etc.; one patient), evidence of a further myocardial infarction (as judged by history and 2nd ECG on day of MRI scan) following the index admission, and prior to MRI scan (two patients). None of the patients had any evidence of Q waves in the baseline (admission) ECG. Magnetic resonance imaging was performed a median of 14 days (inter quartile range 25 days) following the peak in the release of cardiac Troponin 1.

Coronary angiography was performed according to clinical indications either during the same admission or in the subsequent 3 months following admission. The study was approved by our institutional ethics committee and complied with the declaration of Helsinki. Written informed consent was obtained from each patient.

MRI Protocol

Each patient was placed supine in a 1.5-T clinical scanner (Siemens Sonata, Erlangen, Germany), and the integrated spine array combined with a two-channel phased-array receiver coil was placed on the chest for imaging. All images were acquired in end-expiration and were gated to the electrocardiogram. Cine images (True FISP sequence; repetition time 3.0 ms, echo

delay time 1.51 ms and flip angle of 60° were acquired in eight to 10 short-axis views and two long-axis views. The slice thickness was 7 mm, and interslice gap 3 mm. A commercially available Gadolinium-based contrast agent (Gadodiamide, Omniscan®, Nycomed, Amersham, UK) was then administered intravenously at a dose of 0.1 mmol per kilogram of body weight, and contrast-enhanced images were acquired in the same views as those used for cine MRI after a 10-min delay. Contrast-enhanced images were acquired with the use of a segmented Turbo FLASH inversion-recovery sequence that has been described previously (Simonetti et al., 2001). The typical voxel size was $1.9 \times 1.4 \times 7.0$ mm. The inversion time (TI) was modified iteratively to obtain maximal nulling of remote normal left ventricular myocardium with an average value of 330 msecs. Total acquisition time for these postcontrast images never exceeded 25 min. The postcontrast images were coregistered to be the same position as the functional (cineMRI) images by copying the same slice position and by again obtaining the images in end-expiration.

Postprocessing Analysis

For functional analysis, all short-axis slices from the base to the apex were analyzed using Argus cardiac analysis software, version 2002B (Siemens, Erlangen, Germany). The following parameters were determined by planimetry of all the short-axis images in end-diastole and end-systole: LV end-diastolic volume (EDV) (in mL), LV end-systolic volume (ESV) (in mL), LV EF (in %), and LV mass (in grams). Regional wall motion (RWM) was analyzed by an experienced observer blinded to the ECG finding and graded as 0—Normal; 1—Mild or Moderate hypokinesia; 2—severe hypokinesia; 3—akinesia; 4—dyskinesia. Areas of late Gadolinium hyperenhancement (HE) were graded in transmural extent [(0)—no HE; (1)—1–25% HE; (2)—26–50% HE; (3)—51–75% HE and (4)—>75% HE] and quantified using planimetry by a separate, experienced observer who was blinded to the ECG and cine MRI findings. The intraobserver and interobserver variability of planimetry of hyperenhanced tissue in our laboratory is 4% and 7%, respectively.

The 17-segment model as recommended in the recently published American Heart Association (AHA) statement on myocardial segmentation and nomenclature was used for regional wall motion and DE-MRI segmental assessment (Cerqueira et al., 2002). From the short axis images, representative basal (area from the mitral annulus to the tips of the papillary muscles at end diastole), mid-cavity (region that includes the entire length of the papillary muscles), and apical (from area beyond papillary muscles to just before the cavity ends) slices were selected for analysis. The true apex (segment 17), consisting of the apical cap, was evaluated from the vertical and horizontal long-axis images.

Coronary Angiography Protocol

All coronary angiograms were performed according to routine clinical indications. The presumptive culprit lesion in the left anterior descending artery (LAD) was identified and characterized as being either in the proximal (before the 1st septal branch),

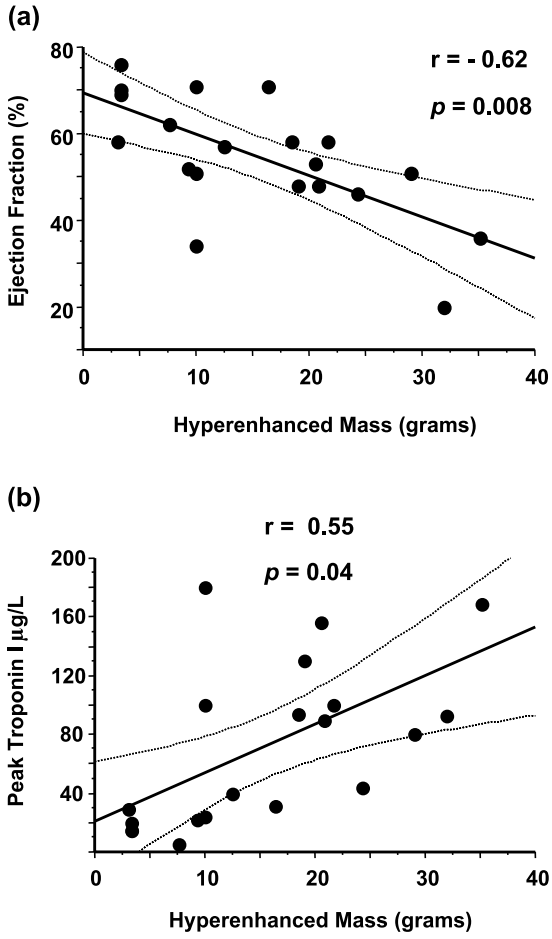

Figure 1. (a) Correlation between the ejection fraction (%) and the mass of hyperenhanced myocardium (grams) in patients studied. (b) Correlation between the peak cardiac Troponin I level (µg/L) and the mass of hyperenhanced myocardium (grams) in patients studied.

middle, and distal segment according to accepted criteria [The National Heart, Lung, and Blood Institute Coronary Artery Surgery Study (CASS)]. All coronary angiograms were evaluated by an experienced cardiologist, who was blinded to the findings of the MRI scan.

Statistical Analysis

Values are expressed as mean (±SD) or median (interquartile range, 25–75%) as appropriate. Continuous variables that were not distributed normally are compared using the Mann-Whitney test, and correlation between such variables is made using the Spearman correlation. All reported p values are two sided, and a value of <0.05 was considered statistically significant.

RESULTS

Patient Characteristics

Mean age of patients was 62±13 years. Seventeen (89%) were male, six (32%) were diabetic, 12 (63%) had hypertension and, 14 (74%) had hypercholesterlaemia. Fifteen patients (79%) had acute reperfusion therapy: thrombolysis in 13 patients and primary percutaneous intervention in two patients. The remaining four patients did not have acute ECG changes that fulfilled criteria for reperfusion therapy.

The mean peak troponin I level was 76±54 μg/L, and the mean left ventricular end-diastolic volume (EDV) was 164±43 mL, end-systolic volume (ESV) was 77±35 mL, and ejection fraction was 53%±16%.

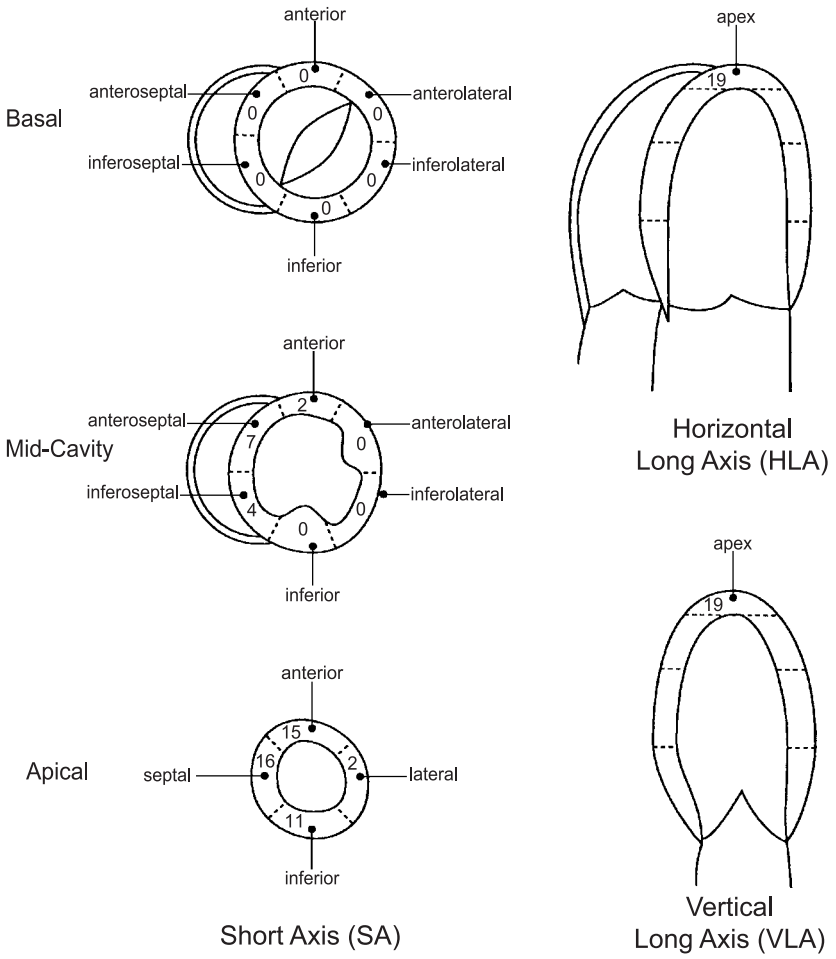

Figure 2. Total number of patients demonstrating any degree of hyperenhancement shown in a 17-segment myocardial model [adapted from Cerqueira et al. (2002)—copyright pending].

Coronary angiography was performed in all 19 patients. Apart from the two patients who underwent primary PCI, the reasons for coronary angiography in the remaining 17 patients were refractory or recurrent ischemia in five patients and myocardial ischemia on provocative testing in 12 patients. The culprit lesion in the LAD was in the middle segment for 14 (74%) patients and in the proximal segment for five (26%) patients.

ECG Results

Eight (42%) patients were classified as showing Minnesota code (MC) 1-1-7 pattern in ECG leads V_1–V_4, eight (42%) patients demonstrated MC pattern 1-2-7, and three (16%) patients demonstrated MC code pattern 1-1-6 in the relevant leads.

Delayed Hyperenhancement MRI

All patients successfully underwent cine and contrast enhanced MRI. Patients were imaged a median of 14 days (4–29 days; 25–75%) days after the index infarction.

All 19 patients had evidence of delayed hyperenhancement in one or more myocardial segments (mean number of affected segments 5.5±2.1). The mean mass of hyperenhanced myocardium was 14±8 grams, or 10±6% of absolute LV mass. There was a significant linear correlation between the mass of hyperenhanced tissue and the ejection fraction ($r = -0.62$; $p = 0.008$; Fig. 1a) as well as a positive correlation between hyperenhanced tissue mass and peak Troponin I ($r = 0.55$; $p = 0.04$; Fig. 1b).

Segment-by-segment analysis, indicating the number of patients affected for each left ventricular segment is shown in Fig. 2. All patients showed evidence of delayed hyperenhancement of the apex (segment 17). This hyperenhancement was 75–100% transmural in 13 patients (68%) and 50–75% transmural in the remaining six patients. In addition, 17 (89%) of patients showed evidence of hyperenhancement in the apical anterior and/or apical septal segments. Seven (37%) and four (21%) patients showed evidence of mid anteroseptal and mid inferoseptal hyperenhancement, respectively, and no patients had any hyperenhancement of basal anteroseptal segments. The transmural extent of hyperenhancement for

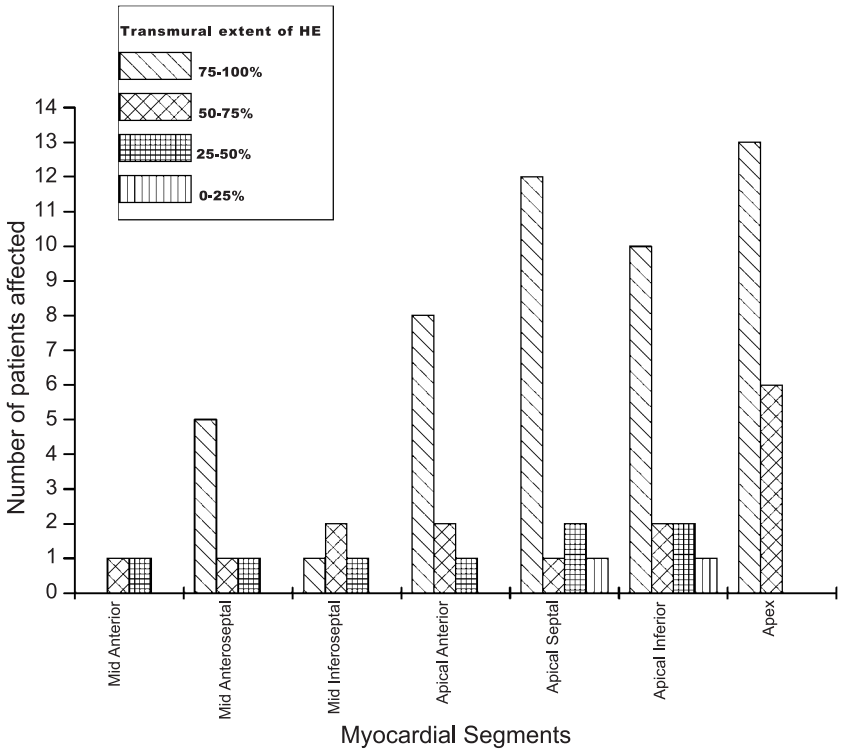

Figure 3. Grade of hyperenhancement in mid cavity and apical myocardial segments according to total number of patients affected.

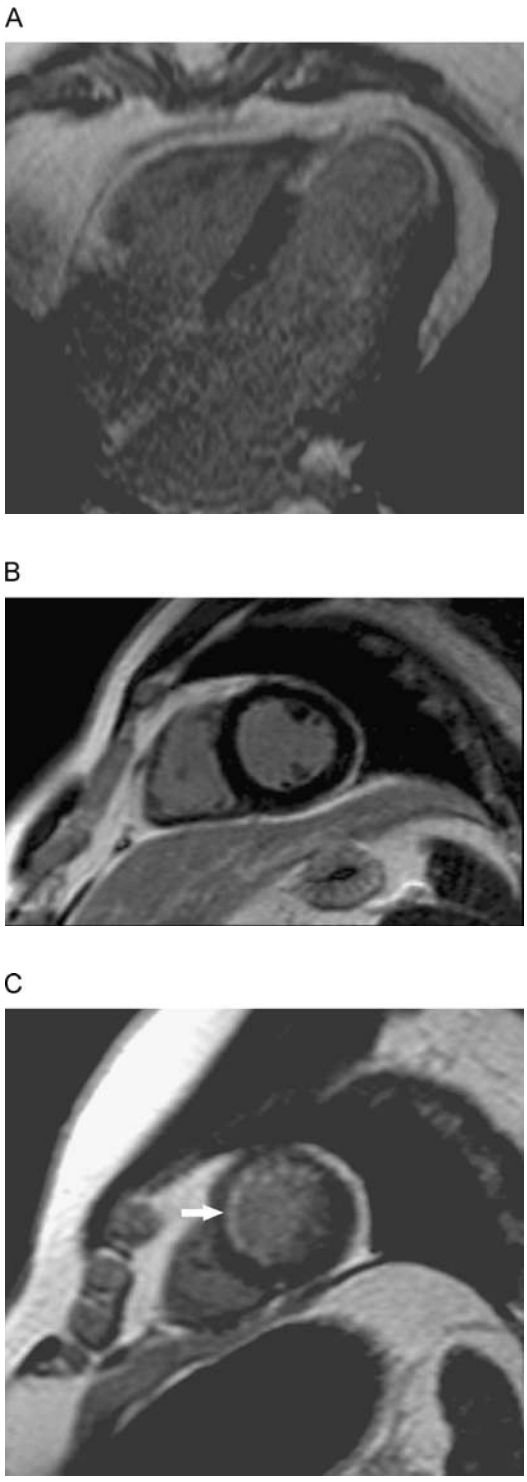

Figure 4. Segmented turbo FLASH (Fast Low Angle Shot) inversion recovery sequence images for a patient in horizontal long axis (A), mid cavity short axis (B), and apical short axis (C) planes showing delayed hyperenhancement involving the apex, and apical septum (arrow) but not the mid cavity anteroseptum.

segments most affected is detailed in Fig. 3 and a patient example is shown in Fig. 4.

Regional Wall Motion

All 19 patients had evidence of regional wall motion abnormalities in one or more segments (mean number of affected segments 7.5 ± 2.3). The apex was the most common segment involved, being affected in all 19 patients, of whom six had akinesia, seven dyskinesia, and six had hypokinesia. The apical anterior and inferior segments were also frequently involved with 15 patients having some degree of regional wall motion abnormality in one or both of these segments. Regional wall motion scores for affected mid cavity and apical left ventricular segments are shown in Fig. 5.

There was a highly significant correlation between the transmural extent of hyperenhancement of a segment and its regional wall motion score when all myocardial segments were analyzed ($r = 0.62$; $p < 0.001$). However, there was poor agreement between the regional wall motion findings in the mid cavity anteroseptum and inferoseptum segments with the corresponding hyperenhancement transmurality in these segments ($r = 0.30$; $p = 0.20$) but excellent correlation between the regional wall motion findings in the apical segments (apical anterior, apical septal, and apex) with their respective transmural grade of hyperenhancement ($r = 0.70$; $p = 0.02$). This discrepancy in the wall motion score and extent of hyperenhancement in the mid cavity myocardial segments indicates that these segments are most likely stunned but not irreversibly injured.

DISCUSSION

The 12 lead electrocardiogram (ECG) remains critical to the diagnosis and localization of acute and healed myocardial infarction (MI) (Turi et al., 1985; Woods and Smith, 1983). Myocardial necrosis is reflected electrocardiographically by QRS negativity and/or a loss of QRS positivity in a lead that is oriented towards the infarcted region (Schamroth, 1993). The traditional electrocardiographic definition of anteroseptal MI is a Q wave or a QS complex >0.03 seconds in leads $V_1 - V_4$ (Chaitman, 2001; Wagner, 2001).

Our finding that all patients presenting with new Q waves in leads $V_1 - V_4$ in the setting of recent, conservatively treated (most received thrombolysis) myocardial infarction had hyperenhancement of the

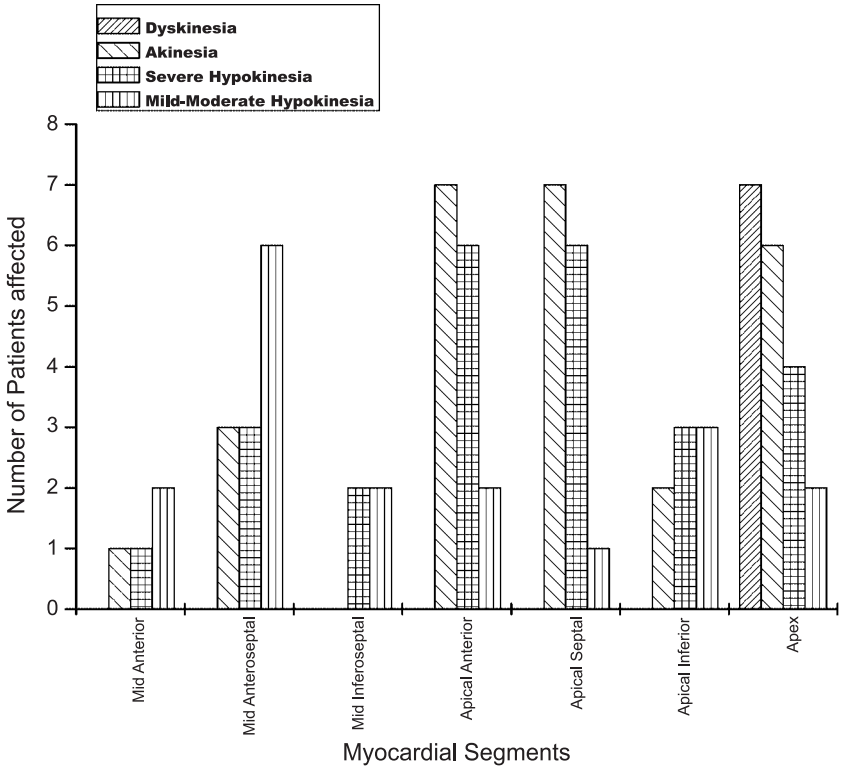

Figure 5. Extent of regional wall motion abnormality in mid-cavity and apical myocardial segments according to total number of patients affected.

apex, and that most (79%) had involvement of the apical anterior and apical septal regions contradicts this traditional dogma. In our study, the mid cavity anteroseptum was only involved in 37% of patients and the basal anteroseptum was never involved. There were no differences in the ECG Q wave pattern that predicted if a patient would have just isolated apical involvement as opposed to those with both apical and mid anteroseptal hyperenhancement.

Our study confirms that most patients presenting with such ECG abnormalities have only a limited amount of irreversibly injured myocardium. However, even though the absolute amount of nonviable tissue is small (mean of 10% of LV mass), it is still sufficient to result in a measurable negative impact on global ejection fraction. Furthermore, our finding of a positive significant correlation between infarcted mass and peak serum Troponin I again underlines the fact that serum Troponin I is a sensitive and specific marker of myocardial tissue necrosis.

Regional wall motion analysis showed differences in the left ventricular apical vs. the mid cavity segments. While the majority of the apical anterior and apical septal segments were akinetic and dyskinetic, most of the patients with mid cavity anteroseptal abnormalities (8/12) were found to have hypokinesia, rather than akinesia or dyskinesia. Moreover, in the 12 patients with mid cavity anteroseptal wall motion abnormalities, hyperenhancement was evident in only seven (58%) patients, indicating that in the rest of these patients these myocardial segments were dysfunctional (stunned or jeopardized ischemic myocardium), but still potentially viable. It is therefore tempting to speculate that earlier studies such as that by Bogarty et al. (who found that 50% of septal areas were also affected) were scoring some viable and not irreversibly damaged anteroseptal regions.

Other aspects of our findings are in agreement with those of Shalev et al. (patients without reperfusion) and Bogarty et al. (patients receiving reperfusion). While these two previous studies using echocardiography/X-ray contrast ventriculography found the apex to be the main segment affected with variable degree of involvement of the septum, we have found, using high resolution delayed enhancement MRI, the apex and apical septal/anterior segments to be principally affected, with infarct extension to the mid cavity anteroseptum in a subset (37%) of patients.

The angiographic findings indicate that the most likely site of the culprit lesion in patients presenting with such ECG abnormalities is in the middle segment of the left anterior descending artery, and distal to origin of the 1st septal branch. This would be in keeping anatomically with the location of the infarction involving the distal septum and the apex. The five patients in whom the culprit lesion was in the proximal LAD segment all showed the delayed hyperenhancement involving the mid cavity anterior and inferior septum in addition to hyperenhancement of the apex and apical septal segments.

Study Limitations

A limitation of the study is that the time from the index event to MRI was a broad range (median 14 days; inter quartile range 25 days). This means that in some patients the scan was performed in the subacute stage after infarction, while in others the images reflect acute infarction. While this might impact on the size of the infarcts observed [with recent questions that DE-MRI may overestimate the size of acute infarction (Ingkanisorn et al., 2002)], this is unlikely to have had an impact on the location of the infarct within the myocardium. Given this broad time range we cannot be absolutely certain that the patients that were scanned later did not have a further (silent) episode of myocardial infarction. Although we attempted to exclude such patients with history and second ECG prior to their MRI scan, both these measure are relatively insensitive and may have missed further small infarcts.

In conclusion, our findings indicate that patients presenting with acute myocardial infarction and new Q waves limited to leads V_1-V_4 should be considered to have principally apical infarction. Our study does not support the notion that these patients have isolated septal or anteroseptal infarction.

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