INTRODUCTION

More than 5.5 million patients visit the emergency room in the United States every year with chest pain; of these, approximately 1.5 to 2 million are admitted to the hospital for acute myocardial infarction (MI). About two-thirds of these MI patients are found to have non-ST-segment elevation MI (NSTEMI, previously referred to as non-Q-wave MI). Previous studies in selected clinical trial populations have reported that up to 15%-20% of patients with NSTEMI undergoing angiography have little or no significant coronary atherosclerosis, suggesting insignificant (ie, non-obstructive) coronary artery disease (CAD). Despite having less significant CAD and small-sized infarcts, patients with NSTEMI have a greater propensity for early recurrence of myocardial ischemia, which may place them at greater risk for recurrent MI and adverse outcomes. The reasons for the apparent dissociation between the severe clinical presentation and trivial angiographic findings remain unclear, but may largely reflect the greater burden of risk factors such as hypertension and its consequences, including left ventricular hypertrophy (LVH).

Recently, there has been growing interest in the contribution of LVH to the clinical expression of myocardial ischemia in patients with hypertension. It has recently been suggested that LVH, whether assessed by ECG or more specifically by echocardiography, may confer a greater risk for MI (particularly NSTEMI) and related adverse events, presumably through obstructive atherosclerotic CAD. It remains unclear, however, whether LVH with or without hypertension contributes independently to acute NSTEMI in patients with non-obstructive CAD, and whether such information is particularly helpful in Blacks who are at higher risk for both uncontrolled hypertension and LVH and the risk of NSTEMI. We therefore sought to investigate the clinical significance and independent contribution of echocardiographic LVH as a correlate of NSTEMI in patients presenting with acute chest pain in a large urban hospital center serving predominantly African American (AA) patients.
We sought to investigate the clinical significance and independent contribution of echocardiographic LVH as a correlate of NSTE-MI in patients presenting with acute chest pain in a large urban hospital center serving predominantly African American (AA) patients.

METHODS

Study Population

We retrospectively selected consecutive patients who underwent cardiac catheterization for evaluation of acute chest pain and suspected myocardial ischemia in the period between January 1995 and December 1996. This analysis included only those patients with normal coronary arteries at cardiac catheterization performed during the same index hospital admission and who also had data available for cardiac markers to confirm the presence or absence of acute NSTE-MI (ie, non-Q-wave MI), as the main outcome. Acute NSTE-MI was diagnosed by positive cardiac markers (defined as serial elevations in CK-MB peaking within 24 hours or CK-MB more than twice the upper limit of normal) among patients admitted with ECG changes including ST-segment depression ≥0.5 mm.9,13 Because ECG criterion can be equivocal in some patients with ECG evidence of LVH, a previous ECG was used for comparison when available to ensure that the observed changes are new.

Cardiovascular Risk Factors

Hypertension was defined as systolic BP ≥140 mm Hg or treatment with antihypertensive medication. Control in medicated hypertensive patients was defined by the number of controlled hypertensives (BP <140/90 mm Hg) divided by the number of treated hypertensives.16 Other traditional risk factors for atherosclerotic CAD (ie, diabetes mellitus, dyslipidemia, obesity, and smoking) were recorded for each patient and defined consistent with national standards.5-4

Echocardiography

Two-dimensional directed M-mode echocardiograms were recorded in standard views,17-19 and measurements were made according to the recommendations of the American Society of Echocardiography. Only echocardiographic examinations performed as close as possible temporal to the index hospital admission for NSTE-MI were used for data analysis. Among those excluded for lack of cardiac marker data, seventeen patients also did not have echocardiographic data available. All remaining patients included in the final study group had completed echocardiographic data available. For purposes of this analysis, patients were grouped according to the presence or absence of LVH as previously reported in our routine clinical practice.19

Coronary Angiography

Cardiac catheterization was performed among patients in the final group for one or more of the following reasons: unstable angina in 36 patients; angina pectoris/exertional chest pain and/or positive stress test/abnormal stress thallium in 33 patients; atypical chest pain in 18 patients; LV dysfunction/heart failure in 22 patients; or recent MI in 22 patients with NSTE-MI. Stress thallium and coronary angiography after the acute event were completed per standard hospital protocols, mostly within the time frame of the index hospitalization before the patients were discharged from hospital. Thus, the present analysis only included procedures performed at the same index hospital admission for this acute NSTE-MI. All angiograms were read by experienced senior attending cardiologists of the hospital.19 Angiograms were considered normal or near normal in the presence of minimal luminal irregularities (ie, a luminal diameter narrowing <10% of any major epicardial coronary vessel or branch). Angiograms showing any signs of plaque formation were not considered for inclusion in the analyses.

Statistical Methods

Data are expressed as mean ± SD unless otherwise specified. Groups were compared for categorical data or frequency of events using the chi-square test and for continuous variables using the Student’s t test for unpaired data. The odds ratios (OR) with their chi-square or Fisher’s exact statistic and their 95% confidence intervals (CI) were used to estimate the relative risk of MI for patients with and without each risk factor. In multivariate analysis, logistic regression was used to identify variables independently associated with acute MI. For this purpose, the results of bivariate analysis also guided the selection of factors included in the multivariate model. All tests were two-sided and a P<.05 was considered statistically significant.

RESULTS

Baseline Characteristics

Of 700 patients included in the chest pain database (Figure 1), we identified 180 (26%) who had angiographically normal coronary arteries or only minimal luminal irregularities. Of these 180 patients, we excluded 49 patients because cardiac marker results were not available in the database. The remaining 131 patients had complete cardiac marker data available at the index hospital admission to confirm the
presence or absence of NSTE-MI and represented the final study group. Table 1 shows the clinical characteristics for the group as a whole and separated by MI status. Overall, mean age was 53 ± 10 years (range 28-82), 56% were women, 76% were Blacks, 19% Caucasians, and 5% Hispanics. Eighty-eight percent had hypertension with 49% uncontrolled as reflected by systolic BP > 140 mm Hg or diastolic BP > 90 mm Hg. Left ventricular hypertrophy was found on echocardiography in 74%. During hospitalization, 22 out of 131 patients (17%) were found to have NSTE-MI by CK-enzyme criteria (serial elevations in CK-MB peaking within 24h or CK-MB > 2 times the upper limit of normal). We found NSTE-MI was common in patients aged 50 to 59 years (36%) and 40 to 49 years (27%). Further examination of selected clinical characteristics by MI status (Table 1) showed that mean systolic BP was significantly higher in patients with NSTE-MI than in the non-NSTE-MI group (156 ± 30 vs 143 ± 25 mm Hg, P < .05). Patients with NSTE-MI were more likely than counterparts without NSTE-MI to be Black (95% vs 72%, P = .02) and less likely to report obesity or use of aspirin prior to the index hospitalization. They were also more likely to have lower LV ejection fraction (EF) on angiography (51 ± 14% vs 59 ± 13%, P < .05) and LV on echocardiography (95% vs 70%, P < .05). We found NSTE-MI was present in 21 out of 97 (22%) patients with LVH compared with 1 out of 34 (3%) patients without LVH (P = .02). Among the 21 NSTE-MI patients with LVH, 4 (19%) had persistent angina suggestive of recurrent myocardial ischemia as confirmed by positive exercise EKG and stress thallium performed during the course of the index hospitalization before discharge (the median length of hospital stay was 7 days). Ten patients with MI (48%) had symptoms of congestive heart failure (HF); EF was much lower among NSTE-MI patients with LVH than counterparts without LVH (49 ± 14% vs 59 ± 13%, P < .05).

Factors Associated with Acute NSTE-MI
To evaluate factors potentially associated with NSTE-MI, we examined all recorded clinical variables (Table 2). With univariate/bivariate analysis, a modestly significant positive correlation was found between NSTE-MI and Black race, elevated systolic BP, and the presence of LVH (Table 2). The results of the multivariable model after controlling for selected variables revealed that these two preexisting medical conditions were the strongest predictors of NSTE-MI; the presence of LVH with OR of 4.00 (95% CI 1.06-10.05) and elevated systolic BP with OR of 3.70 (95% CI 1.01-10.64) (Table 2). Other clinical variables, such as cigarette smoking, cocaine use, diabetes, and history of hypertension or CAD were not predictive of NSTE-MI. Of particular interest, race was no longer significant once LVH and high BP were accounted for in the final model.

DISCUSSION
We report a two-year experience of 700 consecutive patients who were admitted for acute chest pain symptoms at our institution, of whom 22 of 131 (17%) with normal or near-normal angiograms and available cardiac enzymes drawn at the index hospital admission had evidence of myocardial necrosis (ie, NSTE-MI). These findings are of interest in this largely African American (AA) population with a high prevalence of uncontrolled hypertension and echocardiographic evidence of LVH. Our two main findings were:

1) Although no deaths were reported during the index hospitalization, other adverse outcomes were common because more than half presented with either positive exercise EKG and/or stress thallium suggestive of residual myocardial ischemia or showed heart failure symptoms; and

2) Our data indicate that among the clinical parameters commonly available in these patients at the time of initial presentation, echocardiography...
graphic LVH and elevated systolic BP (ie, uncontrolled hypertension) were significantly and independently associated with acute NSTE-MI.

Although other groups have reported an association between LVH and MI in the absence of obstructive CAD, the size of this series and the careful recording of clinical characteristics make this report particularly relevant to hypertensive AA of both sexes and of relatively young age when presenting with acute chest pain syndrome in routine clinical practice.15

Risk Factor Profile
This database describes a subset of adult patients with high rates of angiographically normal or minimally diseased epicardial coronary arteries (26%), who are at a higher risk for NSTE-MI than previously recognized, and may not otherwise be well-represented in large scale cohorts.5,6,7–9 This proportion (26%) is higher than reported (up to 15%) from large scale cohorts of patients with insignificant/non-obstructive atherosclerotic CAD.7–9,20,21

For example, our patients with NSTE-MI had similar age distribution but higher rates of risk factors such as hypertension than those from the PUR-SUIT cohort,7 which did not present data for AA. In that large randomized trial involving 11000 patients with NSTE-acute coronary syndrome (ACS), a sizable proportion (12%, median age <58 years) had insignificant CAD (6% with no CAD and 6% with mild CAD) and a low incidence of adverse outcomes. More recently, the CRUSADE quality improvement initiative provided another large national sample of patients with high-risk NSTE-ACS (n=38,301) presenting to over 400 US hospitals and included 11% Blacks,8 a higher percentage than most earlier published studies. Insignificant CAD (defined as coronary stenoses <50%) was present in 8.6% of patients (n=3,306) and was also associated with low incidence of adverse outcomes compared to patients (n=34,995) with obstructive CAD (>50% stenoses).8 Altogether, these data support the observation that NSTE-MI in the absence of obstructive CAD may be a more significant problem than previously recognized, especially in this group of otherwise young adult patients who are often considered at low risk. The reason for these seemingly variable clinical presentation/outcomes is unclear, but may reflect differences in patient selection or angiographic definitions.22–26 Alternatively, it may be further explained by the fact that in these earlier large clinical trials some

### Table 1. Baseline characteristics and clinical presentation of patients with and without MI

<table>
<thead>
<tr>
<th>Demographics</th>
<th>All (n=131)</th>
<th>No MI (n=109)</th>
<th>MI (n=22)</th>
<th>P*</th>
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<tbody>
<tr>
<td>Age, yrs</td>
<td>53 ± 10</td>
<td>53 ± 11</td>
<td>54 ± 12</td>
<td>.73</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>73 (56)</td>
<td>63 (58)</td>
<td>10 (45)</td>
<td>.35</td>
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<tr>
<td>Black, n (%)</td>
<td>99 (76)</td>
<td>78 (72)</td>
<td>21 (95)</td>
<td>.02</td>
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<tr>
<td>Traditional risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hypertension, %</td>
<td>88</td>
<td>85</td>
<td>91</td>
<td>.73</td>
</tr>
<tr>
<td>Hypercholesterolemia, %</td>
<td>43</td>
<td>49</td>
<td>36</td>
<td>.13</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>34</td>
<td>36</td>
<td>25</td>
<td>.44</td>
</tr>
<tr>
<td>Obesity, %</td>
<td>62</td>
<td>72</td>
<td>20</td>
<td>.04</td>
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<tr>
<td>Tobacco smoking, %</td>
<td>67</td>
<td>65</td>
<td>70</td>
<td>.8</td>
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<td>Cocaine abuse, %</td>
<td>42</td>
<td>36</td>
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<td>Family history of CAD, %</td>
<td>50</td>
<td>52</td>
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<td>Medical treatment</td>
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<td>Hormone replacement therapy, %</td>
<td>9</td>
<td>12</td>
<td>0</td>
<td>.12</td>
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<td>Medications, %</td>
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<tr>
<td>Aspirin</td>
<td>76</td>
<td>84</td>
<td>64</td>
<td>.04</td>
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<tr>
<td>ACE inhibitors</td>
<td>43</td>
<td>39</td>
<td>59</td>
<td>.09</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>26</td>
<td>23</td>
<td>32</td>
<td>.42</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>54</td>
<td>52</td>
<td>59</td>
<td>.64</td>
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<td>Diuretics</td>
<td>38</td>
<td>36</td>
<td>23</td>
<td>.32</td>
</tr>
<tr>
<td>Nitrites</td>
<td>74</td>
<td>81</td>
<td>82</td>
<td>.99</td>
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<td>Lipid lowering agents</td>
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<td>28</td>
<td>18</td>
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<td>Hypoglycemic agents</td>
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<td>18</td>
<td>.78</td>
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<tr>
<td>Clinical findings on presentation</td>
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<td>Uncontrolled blood pressure, %</td>
<td>49</td>
<td>47</td>
<td>62</td>
<td>.24</td>
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<tr>
<td>Blood pressure, mm Hg</td>
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<td></td>
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<tr>
<td>Systolic</td>
<td>145 ± 26</td>
<td>143 ± 25</td>
<td>156 ± 30</td>
<td>.04</td>
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<tr>
<td>Diastolic</td>
<td>84 ± 18</td>
<td>83 ± 19</td>
<td>91 ± 21</td>
<td>.07</td>
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<tr>
<td>Mean</td>
<td>104 ± 20</td>
<td>103 ± 20</td>
<td>113 ± 23</td>
<td>.04</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>61 ± 17</td>
<td>61 ± 15</td>
<td>65 ± 21</td>
<td>.31</td>
</tr>
<tr>
<td>Echocardiographic LVH, %</td>
<td>74</td>
<td>70</td>
<td>95</td>
<td>.03</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>58 ± 13</td>
<td>59 ± 13</td>
<td>51 ± 14</td>
<td>.03</td>
</tr>
<tr>
<td>Plasma cholesterol, mg/dL</td>
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<td></td>
<td></td>
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<tr>
<td>Total cholesterol</td>
<td>211 ± 64</td>
<td>212 ± 58</td>
<td>217 ± 118</td>
<td>.73</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>150 ± 49</td>
<td>155 ± 55</td>
<td>147 ± 29</td>
<td>.74</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>52 ± 25</td>
<td>52 ± 29</td>
<td>65 ± 22</td>
<td>.32</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>176 ± 126</td>
<td>180 ± 131</td>
<td>117 ± 104</td>
<td>.26</td>
</tr>
<tr>
<td>Biochemical markers</td>
<td></td>
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</tr>
</tbody>
</table>
| Peak CK, IU/l | 241 ± 194  | 195 ± 147    | 471 ± 235 | -
| Peak fraction CK-MB, % | 3.69 ± 8.50| .96 ± .97    | 17.08 ± 14.60 | -
| Peak LDH, IU/l | 275 ± 103  | 256 ± 95     | 363 ± 99  | -
| Platelet Counts | 233 ± 79   | 234 ± 84     | 231 ± 90  | .88|

Data are mean ± SD or n and/or %, of patients unless otherwise specified.
MI, myocardial infarction; CAD, coronary artery disease; ACE, angiotensin converting enzyme; LVH, left ventricular hypertrophy; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CK, creatine kinase; LDH, lactate dehydrogenase.

* Overall P for observed differences between the 2 groups of patients with vs without MI.
important, yet modifiable, co-morbid factors such as LVH were not taken into consideration in the analyses. Therefore, further quantifying the relative influence of LVH as an important correlate of NSTE-MI could help identify patients who are more likely to develop this acute coronary event and related complications, thus providing opportunity for early intervention and treatment.\textsuperscript{8,9,26}

relationship between LVH and NSTE-MI

Although our findings substantiate the relative importance of traditional risk factors for CAD including elevated BP/uncontrolled hypertension, we also show that these factors only explain a fraction of the risk of NSTE-MI in the absence of significant CAD; other parameters must contribute as well. In the present study, we chose to investigate the value of echocardiographic LVH as an important predisposing risk factor for NSTE-MI for three reasons. The first reason was that our urban ambulatory and hospital practice serves a very large AA population. It has been proposed that Blacks are more likely than non-Blacks to have a greater prevalence and worse outcomes from atherosclerotic CAD.\textsuperscript{15} It remains unclear however why Blacks have disproportionately higher rates of angiographically normal or insignificantly narrowed (ie, minimally diseased) epicardial coronary arteries despite a higher prevalence of risk factors for atherosclerotic CAD. Another reason was that, we knew based on our own experience\textsuperscript{12,17–19,27,28} and on data from the literature,\textsuperscript{29–32} that AA continue to suffer a disproportionately more virulent course of cardiac end organ damage from hypertension. As many AA patients with hypertension are poorly controlled, not surprisingly there is an increased risk of complications such as LVH. Indeed, epidemiological studies\textsuperscript{29,30} and findings from large autopsy series from our group\textsuperscript{28} suggest that uncontrolled hypertension and LVH contribute most to the excess cardiovascular risk of AAs.\textsuperscript{28–30} The final reason was that there were limited data in the literature regarding the clinical relevance and independent contribution of LVH as a potential correlate of NSTE-MI in patients with insignificant CAD.\textsuperscript{8,9} particularly in Blacks who are at high risk for NSTE-MI and both uncontrolled hypertension and LVH.\textsuperscript{15} Here, we found that LVH, which we previously showed to correlate closely with BP among AA patients with and without hypertension,\textsuperscript{12,17} is also an important independent predictor of cardiac ischemic event (ie, NSTE-MI) in this ethnic group.

Other factors including obesity and associated lipid abnormalities or metabolic disorders such as diabetes (Table 2), previously shown to have a negative impact on the clinical presentation and outcome of these patients, were not predictive of NSTE-MI on multivariate analysis. It is also worth noting that medication usage was comparable between the two study groups and is therefore unlikely to have significantly influenced our results. The same is true for the potential negative influence of smoking\textsuperscript{33} or the use of recreational drugs such as cocaine.\textsuperscript{34}

Possible Explanations for the Adverse Impact of Hypertension and LVH

Among patients with poor BP control, systolic BP was markedly higher in those who eventually had NSTE-MI than in counterparts without

\begin{table}[h]
\centering
\begin{tabular}{|l|l|l|l|}
\hline
Variable & Odds Ratio* & Lower & Upper  \\
\hline
\textbf{Univariate/Bivariate Analysis} & & & \\
Age & 1.008 & 0.966 & 1.051  \\
Male (vs. female) & 1.643 & 0.654 & 4.130  \\
Blacks (vs. non-blacks) & 1.120 & 1.020 & 1.930  \\
History of hypertension & 1.744 & 0.369 & 8.248  \\
Blood pressure: & & & \\
Systolic & 3.970 & 1.026 & 9.164  \\
Diastolic & 1.021 & 0.998 & 1.046  \\
Mean & 1.024 & 1.001 & 1.047  \\
Pulse Pressure & 1.015 & 0.987 & 1.044  \\
Echo-LVH & 4.865 & 1.001 & 12.249  \\
LV ejection fraction & 0.964 & 0.931 & 0.998  \\
Obesity & 0.095 & 0.009 & 0.987  \\
Plasma cholesterol & 1.001 & 0.995 & 1.007  \\
Triglycerides & 0.992 & 0.979 & 1.005  \\
Diabetes mellitus & 0.593 & 0.199 & 1.765  \\
Tobacco smoking & 1.266 & 0.443 & 3.612  \\
Cocaine abuse & 1.350 & 0.211 & 8.619  \\
Family history of CAD & 0.459 & 0.159 & 1.326  \\
Ischemia on thallium & 2.727 & 0.327 & 22.741  \\
\textbf{Multivariate Analysis} & & & \\
Echo-LVH & 4.00 & 1.06 & 10.05  \\
Systolic blood pressure & 3.70 & 1.01 & 10.64  \\
Ischemia on thallium & 4.1 & 1.09 & 13.6  \\
\hline
\end{tabular}
\caption{Factors associated with acute NSTE-MI}
\end{table}

* Odds ratio (OR) are unadjusted estimates from the bivariate analysis. An OR greater than 1 (one) indicates that patients with the characteristics (risk factor) compared to counterparts without the features have a higher odds of having an acute MI; while an OR less than one indicates a lesser odds of having MI.

\textsuperscript{†} P for overall chi square tests that denote a significant OR because the confidence interval does not include 1 (one).

Abbreviations as in Table 1.
NSTEMI. The role of elevated BP or uncontrolled hypertension in predisposing patients to NSTEMI is unclear, but may relate to the integrated influence of cardiac morphologic changes. These early adaptive changes, characterized by elevated LV mass (ie, LVH) in response to high BP as shown in our previous studies, suggest an intermediate stage whereby an incremental increase in BP may predispose individuals to the premature development of atherosclerotic cardiovascular events. The detrimental impact of LVH independent of elevated BP (ie, uncontrolled HTN) on the risk of NSTEMI observed in this study would suggest such a mechanistic link. While the exact mechanisms responsible for this association between LVH and NSTEMI cannot be precisely deduced from the results of the present retrospective registry, preliminary data from our laboratory suggest that LVH may lead to impaired myocardial flow reserve and microvascular dysfunction. In this preliminary study, using dipyridamole myocardial contrast echocardiography to evaluate the transmural distribution of myocardial blood flow (MBF), we found that subendocardial MBF reserve was blunted in hypertensive patients with LVH, suggesting the mechanisms of LVH-induced myocardial ischemia in hypertension. In these patients, symptoms of chest pain typical for angina pectoris may be manifested prior to or at the time of the MI even in the absence of obstructive epicardial CAD. The reasons are possibly related to limited flow reserve and dysfunction of the microvasculature beyond the epicardial coronary arteries which is unable to compensate for increased metabolic and oxygen demand. This could predispose patients to repetitive ischemia within the subendocardial layer that eventually culminates in heart attacks without elevation of the ST segment and subsequent heart failure despite the absence of atherosclerosis or plaques build up in the coronary blood vessels as noted in the present study.

Study Limitations
First, the retrospective nature limited our ability to draw inferences about causal association between BP, LVH and NSTEMI. Because both risk factors were obtained at the same time, we also could not determine their temporal association with adverse in-hospital outcomes. Second, the true prevalence of NSTEMI and related putative risk factors/comorbidities may be considerably underestimated. In retrospect, our data was collected between 1995 and 1996 and might reflect the transition period before the widespread adoption of new definition of MI with the advent of troponin as a more sensitive biomarker for diagnosing acute MI in the mid to late 1990s. Although a more contemporary series of patients might have been relevant to the current practice using troponin, comparing and interpreting the findings in the context of previous clinical studies utilizing the CK-MB criteria would be inappropriate. It is therefore tempting to speculate that troponin T or I, as more sensitive cardiac contractile proteins/markers that allowed for detection of minor myocardial damage with greater specificity, might have classified even a larger number of our patients with NSTEMI in the absence of CAD. Finally, we have not been able to systematically analyze LV mass as a continuous variable and hence determine the magnitude of LVH in patients with MI compared with those who did not experience MI. Instead, for the purposes of this analysis, patients were grouped according to the presence or absence of LVH detected by routine echocardiography. Although this semi-quantitative approach may limit diagnostic sensitivity, specificity, and predictive accuracy, it is unanimously accepted and easy to obtain in routine clinical practice, and has been shown to provide additional information with regard to cardiovascular risk of patient with hypertensive heart disease irrespective of the extent of CAD.

Clinical Implications
Despite the above limitations, our findings may have important clinical implications for the management of NSTEMI in patients with hypertensive heart disease and acute ischemic chest pain syndrome. In this context, the presence of LVH may help identify a subset of patients with NSTEMI and normal coronary arteries who are at particularly high risk for subsequent cardiac events. Perhaps, it is this subgroup of patients who may benefit the most from early aggressive control of predisposing and potentially modifiable factors, such as BP and LVH, by appropriate pharmacological means to reduce the increased risk for both short- and long-term adverse outcomes.

SUMMARY
Echocardiographic LVH and uncontrolled hypertension were found to be independently associated with acute NSTEMI in patients with acute chest pain and coronary arteries that appeared angiographically normal or had only minimal luminal irregularities. While further studies are needed to define the mechanisms underlying this association,
these findings may have implication for management of NSTE-MI in a high-risk subset of adult patients with hypertensive heart disease and insignificant (ie, non-obstructive) CAD.

ACKNOWLEDGMENTS

Many people have contributed to this ongoing research project. We especially thank the staff at the Clinical Research Center at MSM and cardiac catheterization and echo-lab at Grady Memorial Hospital for invaluable technical support.

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REFERENCES


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