Cause of ST Segment Abnormality in ED Chest Pain Patients

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The objective of this study was to determine the electrocardiographic diagnoses of chest pain patients with ST segment elevation (STE) on the 12-lead electrocardiogram (ECG). This study was a retrospective ECG review of adult chest pain patients in a university hospital emergency department (ED) over a 3-month period (January 1, 1996 to March 31, 1996). STE was determined if the ST segment was elevated ≥1 mm in the limb leads and ≥2 mm in the precordial leads in at least two anatomically contiguous leads. Results showed 902 patients who met entry criteria and of whom 202 (22.4%) had STE. Thirty-one (15%) patients had STE acute myocardial infarction (AMI) as the final hospital diagnosis which caused the STE; 171 (85%) patients with STE had non-AMI diagnosis responsible for the ST segment elevation, including left ventricular hypertrophy (LVH) 51 (25%), left bundle branch block (LBBB) 31 (15%), benign early repolarization (BER) 25 (12%), right bundle branch block 10 (5%), nonspecific bundle branch block 10 (5%), left ventricular aneurysm 5 (3%), acute pericarditis 2 (1%), ventricular paced rhythm 2 (1%), and undefined ST segment elevation 35 (17%). Forty-four patients had AMI as the final diagnosis of whom 31 showed STE on presentation to the ED. In 2 of 31 (6%) cases of STE AMI, the ST segment waveform was atypical for acute infarction. We concluded that AMI is not the most common cause of STE in ED chest pain patients. LVH is most often responsible for electrocardiographic STE followed by AMI and LBBB which occur at equal frequencies. (Am J Emerg Med 2001;19:25-28. Copyright © 2001 by W.B. Saunders Company)

The emergency physician (EP) must be an expert in the interpretation of the electrocardiogram (ECG). Patients with chest pain and ST segment elevation (STE) on the 12-lead ECG who are experiencing acute myocardial infarction (AMI) may be candidates for urgent coronary reperfusion via either thrombolysis or primary angioplasty. Such therapy, to maximize benefit, must be delivered as early as possible after the onset of infarction. Other such chest pain patients with electrocardiographic STE may be suffering from a noncoronary (ie, non-AMI) chest discomfort syndrome. The correct identification of these non-AMI patients—both clinically and electrocardiographically—must be made to offer the most appropriate treatments and to avoid potentially dangerous therapies. The following investigation focuses on the various causative syndromes responsible for electrocardiographic STE in the emergency department (ED) chest pain patient.

METHODS

A retrospective review of all adult patients with the chief or secondary complaint of atraumatic chest pain was performed over a 3-month period (January 1, 1996 to March 31, 1996). The study group included all patients admitted to an ED-based chest pain center (CPC). From this group of adult chest pain patients, all individuals with ST segment elevation in two anatomically contiguous electrocardiographic leads meeting the following criteria were used for data analysis: (1) greater than/equal to 1 millimeter (mm) in the limb leads; and (2) greater than/equal to 2 mm in the precordial leads.

The setting of the study was a university hospital ED with an annual patient volume of 60,000 serving a primarily suburban and rural area with an urban section of approximately 40,000 persons; the general population of the area is approximately 120,000. The ED is staffed by emergency medicine resident- and attending-level physicians 24 hours a day. Patients presenting to the ED with atraumatic chest pain who were aged more than 25 years were admitted to the CPC directly from either the triage area or ambulance entrance; additionally, patients aged more than 18 years with atraumatic chest pain and cocaine use were admitted directly to the CPC. Patients with such complaints who were initially admitted to the general ED were also transferred to the CPC. In all cases, the original 12-lead ECG (the study ECG) performed in the ED-based CPC was used for study review.

To determine if STE was present, the study ECG was interpreted by a single investigator retrospectively who measured the ST segments using criteria the following criteria: (1) greater than/equal to 1 mm in the limb leads; and/or (2) greater than/equal to 2 mm in the precordial leads; STE was considered present if either condition was met. For purposes of ST segment measurement, the initial portion of the ST segment was defined as starting at the J point—the juncture point of the QRS complex and ST segment. This phase of the study did not involve ECG interpretation nor determination of the cause responsible for the ST segment elevation. The single electrocardiographic reviewer for the study was a residency-trained/board-certi-

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TABLE 1. Final Hospital Diagnosis for Non-AMI Cases Relative to the Final ECG Interpretation Causing the ST Segment Elevation

Final Hospital Diagnosis	LVH	LBBB	RBBB	NSIVCD	BER	Pericarditis	Paced	Undefined	Aneurysm
Angina/USA	19	19	5	7	7	0	2	1	3
MS pain	19	7	2	3	12	0	0	12	1
Pneumonia/bronchitis	5	4	0	0	3	2	0	11	1
Other	8	1	3	0	3	0	0	11	0
Total	51	31	10	10	25	2	2	35	5

fied emergency physician with additional completion of 3 year internal medicine residency training.

Of those ECGs with ST segment elevation meeting study criteria as noted earlier, the final hospital clinical diagnoses and the initial/final ECG interpretations were recorded. The initial ECG interpretation was considered the interpretation as noted on the ED patient record by the treating physician. The final ECG interpretation—considered the final reading for each study case—was the cardiologist's analysis. A comparison between the initial ED and final hospital ECG interpretations was also made in cases involving ST segment elevation meeting the study criteria. At each data point collection (initial ED ECG interpretation, final hospital ECG interpretation, and final hospital diagnosis), the single reviewer was blinded to the results of the other data fields. For example, on obtaining the final hospital ECG interpretation, the single reviewer was not aware of the initial ED ECG interpretation nor the final hospital diagnosis.

Electrocardiographic diagnoses responsible for the ST segment elevation were recorded; if disagreement was noted between the initial and the final ECG interpretations, the final hospital ECG interpretation was used. In addition, initial and final ECG interpretations were compared.

RESULTS

Nine hundred sixty patients presented to the CPC for chest pain during the study period, representing an approximate 6.4% of the ED population; 58 patients were excluded because of incomplete medical records, leaving 902 patients with the chief or secondary complaint of atraumatic chest pain who were reviewed. Two hundred-two (22.4%) patients showed the above-described ST segment elevation and were used for data analysis. The mean age was 62 years with a range of 26 to 101 years of age and 46% male gender. The average ED census for such a period is 15,000 patients. The prevalence of STE in an ED population is approximately 1.3%.

Thirty-one (15%) patients had AMI as the final hospital diagnosis which was responsible for the ST segment elevation. An additional 13 patients who did not develop ST segment elevation were diagnosed with AMI based on elevations of abnormal serum markers—the non-STE AMI. The following final hospital diagnoses were responsible for the chest discomfort: angina/unstable angina pectoris 63 (37%), musculoskeletal chest pain 56 (33%), pneumonia, bronchitis, and/or pericarditis 26 (15%), and noncardiac chest pain/chest pain of unclear origin/nonspecific chest pain 26 (15%). The diagnoses relative to the electrocardiographic cause of the ST segment elevation are listed in Table 1.

One hundred seventy-one (85%) patients with ST segment elevation had non-AMI diagnosis responsible for the ST segment elevation, including left ventricular hypertrophy (LVH) 51 (25%), left bundle branch block (LBBB) 31 (15%), benign early repolarization (BER) 25 (12%), RBBB 10 (5%), nonspecific bundle branch block 10 (5%), ventricular aneurysm 5 (3%), pericarditis 2 (1%), ventricular paced rhythm 2 (1%), and undefined 35 (17%) (Fig 1). Agreement between the EP and cardiologist electrocardiographic interpretations regarding the cause of STE is depicted in Table 2.

DISCUSSION

The widely recognized benefits of early diagnosis and rapid reperfusion treatment of AMI have only emphasized importance of EP competence in electrocardiographic interpretation. The EP, frequently the initial clinician to evaluate the chest pain patient, is charged with the responsibility of rapid, accurate diagnosis followed by appropriate therapy delivered expeditiously. STE is perhaps the "most demanding" of the electrocardiographic features seen in the chest pain patient; it is "demanding" in that its presence must be explained and, if the cause involves AMI, urgent therapeutic decisions must be made. Although STE is a not uncommon finding on the 12-lead ECG of the chest pain patient, its cause infrequently involves AMI.[1-3] The occurrence of numerous other noninfarctional STE syndromes only reinforces the point that ST segment elevation is an insensitive marker of AMI—and that the importance of correct and rapid interpretation of the ECG is a mandatory skill for the emergency physician.[1] In the instance of the chest pain patient showing STE resulting from a noninfarction syndrome, the correct diagnosis must be made not only to offer appropriate management for that particular illness but also to avoid the incorrect application of potentially dangerous therapies such as thrombolysis.

Approximately 5% of ED visits are for the chief complaint of chest pain.[4-6] Depending on the patient population surveyed, roughly 20% of these patients will be experiencing AMI.[7,8] In the chest pain patient, the rapid and accurate diagnosis of transmural (ST segment elevation) AMI is a formidable challenge for the EP. This diagnosis is accomplished using the 12-lead ECG in conjunction with the history and physical examination; the ECG in this setting also assists the physician in the selection of the proper therapy, in particular the application of treatment aimed at coronary reperfusion.[9] In the appropriate chest pain patient with presumed AMI, ST segment elevation remains the primary criterion for the initiation of thrombolytic therapy, primary angioplasty, and/or other pharmacologic interventions.[10]

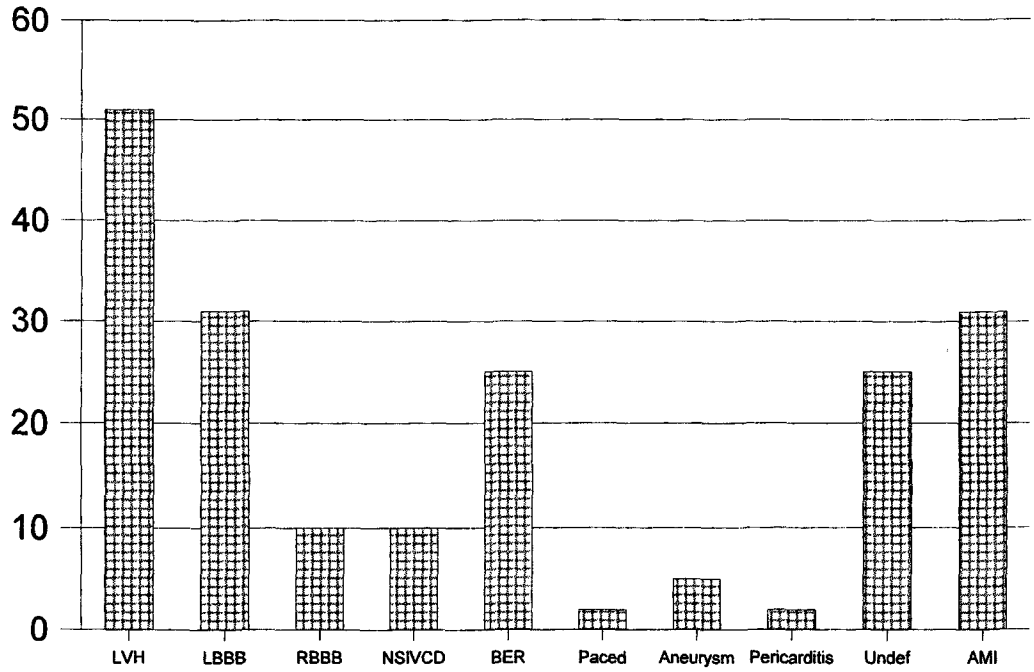

FIGURE 1. Final ECG interpretations causing the ST segment elevation.

ST segment elevation, however, is not a sensitive marker of AMI.[1] In fact, STE must often results from non-AMI electrocardiographic causes.[2] Numerous other noninfarction syndromes which occur in the chest pain patient will manifest STE on the ECG. Certain patterns, such as LBBB, LVH, and left ventricular aneurysm, occur with increased frequency in patients with known coronary artery disease. Other patterns, such as BER and acute pericarditis, are not necessarily associated with ischemic heart disease though they may resemble acute infarction ST segment waveforms. We have reviewed ST segment elevation and its causes extensively elsewhere.[11,12]

TABLE 2. Emergency Physicians' ECG Interpretation Concerning ST Segment Elevation Relative to the Cardiologist Interpretation (Considered as the Standard for Diagnosis)

ECG Syndrome (Cause of ST Segment Elevation)	EM Interpretation (No. Correct/No. Total of Same Reading) [% Correct]
Aneurysm	3/5 [60]
NSIVCD	8/10 [80]
BER	22/25 [90]
AMI	29/31 [94]
LVH	49/51 [96]
LBBB	30/31 [97]
RBBB	10/10 [100]
Pericarditis	2/2 [100]
Paced	2/2 [100]

The existing literature[2,3] suggests that AMI is not the most common cause of STE among patients with chest pain—such is true in the prehospital setting[2] as well as the coronary care unit.[3] The results of this study in the ED found similar results. LVH was the primary cause of STE in the adult ED chest pain patient. LBBB and AMI produced similar rates of STE in this patient population after the LVH pattern. One prehospital study of adult chest pain patients showed that the majority of patients manifesting STE on the ECG did not have AMI as a final hospital diagnosis; LVH and LBBB accounted for the majority of the cases.[2] Furthermore, Miller et al[3] showed that in patients admitted to the coronary intensive care unit with presumed AMI, STE was diagnostic for acute infarct in only half of patients with a past history of ischemic heart disease; left ventricular aneurysm as well as other such STE, non-AMI syndromes were responsible for the ST segment abnormalities resembling acute infarction in this patient group.

The American College of Cardiology/American Heart Association Guidelines for the Management for Patients with Acute Myocardial Infarction[13,14] consider the presence of electrocardiographic ST segment elevation of greater than 0.1 mV in two anatomically contiguous leads a class I indication for urgent reperfusion therapy in the patient presumed to have AMI. Interestingly, these Guidelines do not address the various syndromes potentially responsible for electrocardiographic STE in the chest pain patient. Rather, they mandate urgent reperfusion therapy in the presumed AMI patient with two anatomically oriented leads demonstrating greater than 0.1 mV of elevation.[13,14] Clearly, in this patient subset, such a mandate would have resulted in many unnecessary, potentially dangerous applications of primary reperfusion treatments. Obviously, many non-AMI syndromes are recognized as such by the EP using clinical,

examination, and electrocardiographic clues, thereby providing the most appropriate ED management.

Another recent policy statement addressing treatment considerations in the ED chest pain patient—the American College of Emergency Physicians' *Clinical Policy: Critical* Issues in the Evaluation and Management of Adult Patients Presenting with Suspected Acute Myocardial Infarction or Unstable Angina[15]—speaks to this electrocardiographic diagnostic issue. The Policy states ". . . ST-segment elevations greater than 0.1 mV in 2 or more contiguous leads that are not characteristic of early repolarization or pericarditis, nor of a repolarization abnormality form LVH or BBB . . ." are candidates for fibrinolytic therapy consideration in the ED.[15] Such a statement is much more comprehensive, addressing the reality of the situation much more appropriately than the ACC/AHA publications,[13,14] particularly if one considers the results of this study.

CONCLUSIONS

ST segment elevation less often results from AMI among adult chest pain patients in the ED. LVH is the most frequent pattern followed by AMI and LBBB, occurring at equal frequencies. EPs must consider the various causes of STE in the chest pain patient, realizing that not all such instances of elevation represent myocardial infarction. Educational efforts should be directed towards the correct recognition of the various causes of STE—both AMI and non-AMI origins—so that the correct therapies may be delivered in appropriate fashion.

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