Errors in Emergency Physician Interpretation of ST-segment Elevation in Emergency Department Chest Pain Patients

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Abstract. **Objective:** To determine the rate of error in emergency physician (EP) interpretation of the cause of electrocardiographic (ECG) ST-segment elevation (STE) in adult chest pain patients. **Methods:** The authors conducted a retrospective ECG review of adult chest pain patients in a university hospital emergency department (ED) over a three-month period (January 1 to March 31, 1996). ST-segment elevation was determined to be present 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. Initial EP ECG interpretation was compared with the final interpretation by a cardiologist supported by the results of various clinical investigations. The rate of incorrect ECG diagnosis was calculated. **Results:** Two hundred two patients had STEs. The rate of ECG STE misinterpretation was 12 of 202 (5.9%). The most frequently misdiagnosed form of STE was left ventricular aneurysm, for which two of five cases were believed to represent acute myocardial infarction (AMI). The benign early repolarization (BER) pattern was the second most frequently misinterpreted STE entity—in a total of three cases, two were initially noted to represent pericarditis and one AMI. ST-segment elevation resulting from actual AMI was initially incorrectly noted to be noninfarction in etiology in two cases, one patient with BER and the other with left ventricular hypertrophy. **Conclusions:** Emergency physicians show a low rate of ECG misinterpretation in the patient with chest pain and STE. The clinical consequences of this misinterpretation are minimal. **Key words:** electrocardiogram; ST-segment elevation; medical errors; emergency department. ACADEMIC EMERGENCY MEDICINE 2000; 7:1256–1260

THE emergency physician (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.[1] This rapid, accurate diagnosis assumes EP competence in electrocardiographic interpretation. The widely recognized benefits of early diagnosis and rapid reperfusion therapy of acute myocardial infarction (AMI) have only emphasized the need for this mastery of the electrocardiogram (ECG). Electrocardiographic ST-segment elevation (STE) in the chest pain patient may be misinterpreted as AMI when, in fact, a nonfarction etiology is responsible; conversely, a diagnosis of a noninfarction cause of the STE may be made in the setting of actual AMI. In either instance, appropriate emergency department (ED) therapy and disposition would be possible only if the correct clinical and electrocardiographic diagnoses are made. Numerous noninfarction electrocardiographic syndromes encountered in the chest pain patient may manifest STE on the ECG. Certain patterns, such as left bundle branch block (LBBB), left ventricular hypertrophy (LVH), and left ventricular aneurysm (LVA), occur with increased frequency in patients with known coronary artery disease; these patterns may confound the ED evaluation by mimicking AMI with STE on the ECG. Other patterns, such as benign early repolarization (BER) and acute pericarditis (AP), are not necessarily associated with ischemic heart disease, though they may resemble acute infarction ST-segment waveforms.

One out-of-hospital study of adult chest pain patients demonstrated that the majority of patients manifesting STE on the ECG did not have AMI as a final hospital diagnosis; LVH and LBBB followed by other syndromes such as BER accounted for the majority of the cases.[2] Among adult ED chest pain patients, STE was encountered in 22% of cases. Acute myocardial infarction infrequently was the cause of this STE and was the final hospital diagnosis in only 15% of this population. Benign early repolarization was encoun-

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tered almost as often as AMI (13%).[3] Furthermore, Miller et al.[4] demonstrated 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; LVA as well as other such STE, non-AMI syndromes was responsible for the ST-segment abnormalities resembling acute infarction in this patient group.

These syndromes causing STE not related to AMI are not infrequently misdiagnosed as acute infarction, which then may subject the patient to unnecessary and potentially dangerous therapies and procedures. For example, a report by Sharkey et al. noted that 11% of patients receiving thrombolytic agent were not experiencing AMI. The electrocardiographic syndromes producing this pseudo-infarct STE included BER (30%), LVH (30%), and various intraventricular conduction abnormalities (30%).[5] Misinterpretation of electrocardiographic STE has been reported; at times, these errors in interpretation have significantly impacted medical care.[5–8]

This study was undertaken to determine the rate of incorrect electrocardiographic interpretation of STE in the ED chest pain patient by the EP.

METHODS

Study Design. A retrospective review of all adult patients with the chief complaint of atraumatic chest pain and electrocardiographic STE was performed over a three-month period (January 1 to March 31, 1996). The study was reviewed by the institution's internal review board and was considered exempt from informed consent due to its retrospective nature.

Study Setting and Population. 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 chest pain center (CPC) manages an annual volume of 4,000 patients who are ED patients, representing approximately 7% of the general ED annual census. The ED is staffed by emergency medicine (EM) resident- and attending-level physicians 24 hours a day. The CPC is located within the ED; patients in the CPC are under the direct supervision of the ED attending physician.

The study group was drawn from patients admitted to the ED-based CPC with the chief complaint of chest pain. From this group of adult chest pain patients, all individuals with STE in two anatomically contiguous electrocardiographic leads were used for data analysis.

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 the 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. Only patients with chest pain as the chief complaint were entered in the study. In all cases, the original 12-lead ECG performed in the CPC was used for study review.

Measurements. Electrocardiographic STE was determined if the ST-segment morphology met the following criteria, which were used for data analysis: 1) $\geq$1 millimeter (mm) in the limb leads; and 2) $\geq$2 mm in the precordial leads. In either case, the STE must have been present in at least two anatomically contiguous leads. The determination of STE was made retrospectively using the previously noted criteria by the authors who were blinded to the cardiologist's interpretation of the ECG, the clinical diagnosis, and the results of any diagnostic studies.

In all cases, the initial ECG was interpreted retrospectively by the authors who reviewed the 12-lead ECG and measured the ST segments. Of those ECGs with STE meeting study criteria as noted above, the final hospital clinical diagnoses and the initial/final ECG interpretations were recorded. The final ECG interpretation—considered the final reading for each study case—was the cardiologist's analysis supported by clinical investigations when appropriate. A comparison between the initial ED and final hospital ECG interpretations was also made in cases involving STE meeting the above criteria. At each data point collection (initial ED ECG interpretation, final hospital ECG interpretation, and final hospital diagnosis), the reviewers were blinded to the results of the other data fields. For example, upon obtaining the final hospital ECG interpretation, the reviewers were not aware of the initial ED ECG interpretation.

For this study's purposes only, an initially incorrect electrocardiographic interpretation was determined if: 1) the reading was listed as "*entity* vs *entity*," i.e., no specific diagnosis was made and all listed were incorrect; 2) an incorrect reading was the sole reading; or 3) initial treatment decisions were documented/initiated for the incorrect diagnosis, i.e., a thrombolytic agent was ordered for a patient with chest pain and BER on the ECG.

Data Analysis. Electrocardiographic diagnoses responsible for the STE were recorded; if disagreement was noted between the initial and the final ECG interpretations, the final hospital ECG inter-

TABLE 1. Emergency Physicians' (EPs') Electrocardiogram (ECG) Interpretation Concerning ST-segment Elevation Relative to the Cardiologist Interpretation (Considered to Be the Standard for Diagnosis)

ECG Syndrome (Cause of ST-segment Elevation)	EP Interpretation (Number Correct/Number Total of Same Reading) [% Correct]
Aneurysm	3/5 [60%]
NSIVCD*	8/10 [80%]
Benign early repolarization	22/25 [90%]
Acute myocardial infarction	29/31 [94%]
Left ventricular hypertrophy	49/51 [96%]
Left bundle branch block	30/31 [97%]
Right bundle branch block	10/10 [100%]
Acute pericarditis	2/2 [100%]
Paced rhythm	2/2 [100%]

*NSIVCD = nonspecific intraventricular conduction delay.

pretation was used. In addition, initial and final ECG interpretations were compared.

RESULTS

Two hundred two patients had STEs. The rate of electrocardiographic STE misinterpretation was 12 of 202 (5.9%). The most frequently misdiagnosed form of STE was BER in three cases; two were initially noted to represent pericarditis and one was diagnosed as an AMI. The LVA pattern was the second most frequently misinterpreted STE entity, with two of a total of five cases thought to represent AMI. ST-segment elevation resulting from actual AMI was initially incorrectly noted to be noninfarction in etiology in two cases, one patient with BER and one with LVH. Agreement between the EP and cardiologist electrocardiographic interpretations regarding the cause of STE is depicted in Table 1. Table 2 lists the incorrect initial ECG diagnoses relative to the final, correct diagnosis. No patient without the ultimate diagnosis of AMI who was initially misdiagnosed received acute revascularization therapy.

DISCUSSION

When initial misdiagnoses were investigated in our study, several concerning findings were noted. The initial incorrect interpretation of AMI in noninfarction situations was encountered; the misidentification of non-AMI when myocardial infarction was, in fact, present was also seen. The most frequently misdiagnosed form of STE in this study population was LVA, for which two of 5 cases were believed to represent AMI. Upon additional clinical investigation, other noninfarction diagnoses were made; undoubtedly, the review of either previous ECGs or past medical records provided the important clinical information that enabled the EPs to arrive at the correct diagnosis; similar rates of such misidentification have been described elsewhere with aneurysmal STE.[5]

The BER pattern was the second most frequently misinterpreted STE entity—in a total of three cases, two were initially noted to represent pericarditis and one AMI. The incorrect diagnosis of BER as pericarditis, while troublesome, did not subject the patients to significant, unnecessary therapeutic maneuvers. More concerning, the initial misdiagnosis of AMI in the BER patient could certainly subject the patient to not only unnecessary admission but also unwanted, potentially dangerous therapies such as thrombolysis; fortunately, none occurred in this review. Concerning BER, these diagnostic difficulties have been encountered in other instances. For example, Sharkey et al.[5] has noted a 30% rate of incorrect thrombolysis among patients with non-AMI STE who incorrectly receive a thrombolytic agent; furthermore, the electrocardiographic distinction between acute myopericarditis and BER is notoriously difficult at times.[9]

ST-segment elevation resulting from actual AMI was initially incorrectly noted to be noninfarction in etiology in two cases. In both instances, the initial, upsloping portion of the ST segment was concave, suggesting a noninfarction cause of the ST-segment waveform abnormality. One patient was thought to have BER on the ECG, while the second was incorrectly noted to have LVH-related STE. In most cases of AMI, the initial upsloping portion of the ST segment usually is either convex or flat; if the STE is flat, it may be either horizontally or obliquely so. Conversely, concave STE suggests a noninfarction cause of the waveform abnormality. An analysis of the ST-segment waveform may be particularly helpful in distinguishing among the various causes of STE and identifying the AMI case. This technique uses the morphology of the initial portion of the ST segment/T wave. This portion of the cardiac electrical cycle is defined as beginning at the J point and ending at the apex of the T wave. The use of this STE waveform analysis in ED chest pain patients increased the sensitivity and positive predictive value for correct electrocardiographic diagnosis of AMI markedly.[3] This morphologic observation should be used only as a guideline. As with most guidelines, it is not infallible; patients with STE due to AMI may have concavity of this portion of the waveform.[9]

Left ventricular hypertrophy and LBBB produced STE, which initially suggested the electrocardiographic diagnosis of AMI. In this study population, LVH was misinterpreted as AMI in two

TABLE 2. Incorrect ST-segment Elevation Diagnoses Relative to Correct Electrocardiogram (ECG) Diagnosis

ECG Syndrome* (Initial, Incorrect Interpretation)	Total Incorrect Interpretations	ECG Syndrome* (Final, Correct Interpretation)				
		AMI	LVH	LBBB	BER	Pericarditis
AMI	2	—	1	0	1	0
LVH	2	2	—	0	0	0
LBBB	1	1	0	—	0	0
Aneurysm	2	2	0	0	0	0
BER	3	1	0	0	—	2

*AMI = acute myocardial infarction; LVH = left ventricular hypertrophy; LBBB = left bundle branch block; BER = benign early repolarization.

cases and LBBB as AMI in one case. These two patterns are well known to hinder the diagnosis of AMI via ECG—both as masquerading and obscuring factors—and have been noted to cause similar diagnostic confusion in other situations.[5,9]

Emergency physicians are forced to interpret the ECG in real time, while therapy and additional evaluation is in progress. Such electrocardiographic interpretation frequently occurs without either the benefit of previous ECGs for comparison or the results of additional clinical investigations. Despite this less-than-optimal atmosphere, the EPs in this study interpreted the ECG with respect to the STE incorrectly in 5.9% (12 instances of error among 202 cases of STE) of cases relative to the cardiologist. The cardiologist frequently performs such a review after the initial evaluation has occurred and with the benefit of prior ECGs and the results of other studies.

Fortunately, in this study population, these EP errors in electrocardiographic interpretation did not translate into significant therapeutic mistakes. These syndromes causing STE not related to AMI are not infrequently misdiagnosed as acute infarction, which then may subject the patient to unnecessary and potentially dangerous therapies and procedures. For example, a report by Sharkey et al. noted that 11% of patients receiving a thrombolytic agent were not experiencing AMI. The electrocardiographic syndromes producing this pseudo-infarct STE included BER (30%), LVH (30%), and various intraventricular conduction abnormalities (30%).[5] This incorrect electrocardiographic interpretation by EPs has been noted in other reviews as well.[6–8]

The American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the management of patients with AMI[10,11] consider the presence of electrocardiographic STE of >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 >0.1 mV of elevation.[10,11] 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 on the evaluation and management of patients with suspected AMI or unstable angina[12]—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.[12] Such a statement is much more comprehensive, addressing the reality of the situation much more appropriately than the ACC/AHA publications,[10,11] particularly if one considers the results of this study.

LIMITATIONS AND FUTURE QUESTIONS

This study is limited by several issues, including its retrospective study design, patient identification methods, nonstandardized documentation of electrocardiographic diagnosis, and single-institution base for the patient population. The study's retrospective nature introduces the potential for significant bias and other methodologic errors. Patients were entered into the study if they presented with chest pain and manifested STE according to preset criteria. This STE determination, for study purposes, was determined retrospectively; such determination may introduce error into the study. The lack of a criterion standard diagnostic method for the various ECGs represents yet another significant source of error in this study.

The major future question regarding the mis-

interpretation of electrocardiographic STE by EPs involves methods aimed at reducing this incorrect diagnosis. A survey[13] of EPs investigating their ability to diagnose correctly the cause of electrocardiographic STE revealed that the most frequently misinterpreted patterns were LVA, AMI with atypical STE morphology, BER, and AP. In certain cases, the misinterpretation led the EP to initiate inappropriate therapy, including thrombolysis. Educational programs aimed at EP instruction regarding electrocardiography may improve the EP's correct diagnostic rate. Perhaps structured educational programs, both within EM residency educational programs and in continuing medical educational conferences, may have an impact on the correct diagnosis by EPs. Future determinations of an individual EP's competency in electrocardiographic interpretation may identify clinicians with diagnostic difficulties; focused educational programs in the problem areas may further reduce the error rate.

CONCLUSIONS

The initial misdiagnosis of the various STE syndromes occurs infrequently and, in this study, did not appear to alter therapy in significant form—both the inclusion of unnecessary treatment and the provision of inappropriate therapies. Left ventricular aneurysm is the most frequently encountered form of STE that is incorrectly diagnosed as AMI. Emergency physicians 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 toward the correct recognition of the various causes of STE—both AMI and non-AMI etiologies—so that the correct therapies may be delivered in appropriate fashion. The EP interprets the ECG with respect to STE correctly in most cases.

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