**Objective:** To investigate the specificity of the electrocardiographic diagnosis of ST-segment elevation myocardial infarction in the critical care unit setting.

**Design:** Retrospective observational cohort analysis.

**Setting:** An 880-bed tertiary care teaching hospital with 120 intensive care unit beds.

**Patients:** The population included medical, surgical, trauma, and neurosurgical intensive care unit patients.

**Interventions:** Electrocardiograms were systematically collected to include all consecutive recordings over a 15-month period in which the interpretation software indicated ***ACUTE MI***. Patient demographics, markers of intensive care unit complexity, and hospital mortality were ascertained. The electrocardiograms were then further evaluated by a blinded, board-certified cardiologist for agreement or disagreement with the interpretation software. Serum troponin measurements obtained within 96 hrs of electrocardiogram acquisition were used to determine the likelihood of myocardial infarction.

**Measurements and Main Results:** Over the 15-month study period, the interpretation software diagnosed ST-segment elevation myocardial infarction in 67 of 2243 intensive care unit patients (2.99%) who had an electrocardiogram performed. In the final study population of 46 cases with electrocardiographic ST-segment elevation myocardial infarction, 85% had peak troponin elevation <5 ng/mL, a strong suggestion against clinical ST-segment elevation myocardial infarction. The cardiologist agreed with the computer interpretation in 39% (18 of 46) of cases, but of those 18 patients, only six showed a significant rise in the troponin level. The cardiologist disagreed with the computer interpretation in 60.9% (28 of 46) of cases and of those, one patient had a marked elevation of the cardiac troponin.

**Conclusions:** ST-segment elevation myocardial infarction in the intensive care unit is a relatively common electrocardiographic reading both by standard interpretation software and by expert evaluation. In contrast to nonintensive care unit patients who present with chest pain, the electrocardiographic ST-segment elevation myocardial infarction diagnosis seems to be a nonspecific finding in the intensive care unit that is frequently the result of a variety of nonischemic processes. The vast majority of such patients do not have frank ST-segment elevation myocardial infarction. (Crit Care Med 2010; 38:2304–2309)

**Key Words:** myocardial infarction; electrocardiogram; troponin; critical care; intensive care unit

*See also p. 2412.

Cardiology Fellow (SLR), Medical College of Virginia, Richmond, VA; Fellow (JH), Pulmonary & Critical Care Medicine, Department of Internal Medicine, University of Birmingham-Alabama, Birmingham, AL; Associate Program Director (MWH), Department of Internal Medicine, Carolinas Medical Center, Charlotte, NC; Director of Biostatistics (HJN), Carolinas Medical Center, Charlotte, NC; Professor of Medicine (LL), UNC Chapel Hill, Charlotte, NC; and Chief (LL), Division of Cardiology, Department of Internal Medicine, Carolinas Medical Center, Charlotte, NC.

For information regarding this article, E-mail: Laszlo.Littmann@carolinashealthcare.org

Copyright © 2010 by the Society of Critical Care Medicine and Lippincott Williams & Wilkins

DOI: 10.1097/CCM.0b013e3181fa02cd
Anecdotal observations suggest that in intensive care units (ICUs) the electrocardiographic (ECG) finding of ST-segment elevation myocardial infarction (STEMI) is a relatively common occurrence. An ECG diagnosis of STEMI raises the urgent question whether these typically critically ill patients should be considered for immediate reperfusion. Outside the ICU setting there is little confusion or debate about the use of the ECG in patients who present with chest pain. The ECG is the initial branch point in the decision tree to evaluate patients with acute chest pain because the presence of ST segment elevation usually signifies occlusive thrombus necessitating immediate reperfusion (1–4). ICU patients, however, are typically poor candidates for thrombolytic therapy or emergent cardiac catheterization with balloon angioplasty and stenting. It is imperative, therefore, to understand the clinical significance of electrocardiographic STEMI findings in critically ill patients.

Numerous chronic ECG abnormalities and chronic or acute disease processes can cause ST-segment elevation that is not the result of STEMI (5). These include pericarditis, myocarditis, pulmonary embolism, hyperkalemia, left ventricular hypertrophy, left bundle branch block, and benign early repolarization (5). Additionally, in acutely ill patients, case reports and brief case series have described ST segment elevation in association with severe hyperkalemia (6), high-dose propofol (7), electric cardioversion (8), fulminant hepatic failure (9), atelectasis (10), tension pneumothorax (11), pancreatitis (12), pneumomediastinum (13), hypercalcemia (14), and the apical ballooning syndrome (15). Our study was undertaken to systematically collect ECGs of critically ill patients in the ICU setting with ECG diagnostic criteria of STEMI, and to use the cardiac serum marker, troponin, to either confirm or refute STEMI as a likely clinical diagnosis. Participants in this CME activity should be better able to evaluate the value of ECG interpretations and troponin measurements in the diagnosis of STEMI for ICU patients. Participants also should be better able to assess the need for emergency cardiac catheterization in this population.

**MATERIALS AND METHODS**

**Patient Selection.** This was a retrospective cohort study conducted at Carolinas Medical Center, an 880-bed tertiary care teaching hospital with 120 ICU beds. The study included medical, surgical, trauma, and neurosurgical ICU patients. Also included were cardiac ICU patients admitted for a diagnosis other than acute coronary syndrome. All ECGs were recorded using GE-Marquette ECG carts (Milwaukee, WI) fitted with the 12SL interpretation software. Initial patient inclusion criteria were determined by the interpretation software. The ECGs were systematically collected by searching the Marquette Universal System for Electrocardiography ECG database (Hartford Hospital, Hartford, CT) in a retrospective manner to include all consecutive ECGs over a 15-month period from January 2006 through April 2007 in which the interpretation software indicated "***ACUTE MI***. Patients who did not have at least two concomitant serum troponin measurements were excluded from the final database. The study was approved by Carolinas Medical Center’s Institutional Review Board, which, because of the retrospective nature of the study, waived the need for informed consent.

**Laboratory Studies.** The serum cardiac troponin I assay was a two-site immunoenzymatic sandwich method. The reference ranges at our institution are as follows: normal, <0.07 ng/mL; borderline, 0.07–0.5 ng/mL; and abnormal, >0.5 ng/mL. Before data acquisition, an arbitrary serum troponin measurement of <5 ng/mL was used as the cutoff value to indicate that the clinical diagnosis of STEMI was unlikely. This cutoff value was chosen based on two sets of data. First, in 265 consecutive cases with proven STEMI at our institution during the same time period of this study, the mean serum troponin was 63.6 ± 103.84 ng/mL. Second, in a recent study of 82 patients with proven acute myocardial infarction, the mean troponin elevation was 17.17 ± 3.74 ng/mL at 6 hrs, and it continued to rise at 48 hrs (16). Based on these data, we felt that using a peak troponin level of <5 ng/mL would reliably exclude the clinical diagnosis of STEMI.

**ECG Interpretation.** Initial interpretation was performed by the GE-Marquette 12SL interpretation software. The ECGs were further evaluated by a board-certified cardiac electrophysiologist, an expert ECG interpreter, who was blinded to all clinical data as well as to the purpose and scope of the study. The blinded cardiologist had only the ECG recordings, no clinical background, and was asked to simply agree or disagree with the computer interpretation of STEMI.

**Data Collection.** Using Careline and CHAMP Portal patient databases, patient demographics, ICU complexity (use of pressors, mechanical ventilation, hemodialysis), laboratory analysis (complete blood counts and serum electrolytes), and hospital mortality were ascertained. Serum troponin measurements were obtained within 96 hrs of ECG acquisition. All patients had at least two troponin determinations including 15 patients who had three or more troponin tests done. Of the multiple serum troponin measurements, the highest value was always chosen.

**Statistical Analysis.** Descriptive statistics including means and sds, or counts and percents were calculated. For data measured on the interval scale, the Student’s t test was used to compare the means of two groups of patients. For data measured on the nominal scale, the chi square test or the Fisher’s exact test was used to compare proportions between the groups. For data measured on the ordinal scale, or if the data were not normally distributed, the Wilcoxon rank sum test was used. A two-tailed p value of <.05 was considered statistically significant. The SAS software, Version 9.1 (SAS Institute Inc, Cary, NC), was used for all analyses.

**RESULTS**

Over the 15-month study period, 2243 ICU patients had at least one ECG recorded. Of those, 67 patients (2.99%) had a computer interpretation software diagnosis of STEMI. Fourteen cases were excluded because the reason for patient transfer or admission to the ICU was for likely acute coronary syndrome, and seven patients did not have concomitant serum troponin measurements. After these exclusions, the final study population included 46 cases with STEMI diagnosed by the ECG interpretation software. Of these, 39 patients (84.8%) had serum troponin levels <5 ng/mL, whereas seven patients (15.2%) had troponin levels ≥5 ng/mL (Table 1). Of the 39 patients with troponin <5 ng/mL, only 12 had troponins in the normal range. Twenty patients had borderline troponin elevations, whereas seven patients had abnormal peak troponin levels but <5 ng/mL (mean, 1.93 ng/mL; range, 0.78–4.83 ng/mL). In the seven patients with troponin ≥5 ng/mL, the mean troponin level was 14.1 ng/mL with a range of 5.92–36.88 ng/mL. There were no statistically significant differences between the groups of patients with troponin levels <5 ng/mL or ≥5 ng/mL with regard to prehospital demographics, ICU complexity (use of pressors, ventilatory status, dialysis), laboratory findings (electrolytes, complete blood counts), or mortality, but there was a suggestion of higher mortality and more frequent use of inotropes in the high-troponin group (Table 1). The small absolute number of cases, however, questions the statistical and clinical relevance of these findings.

The ECGs were also evaluated by a blinded board-certified cardiac electro-
Of cases and of those, one patient had marked elevation of the cardiac troponin. Using the troponin elevation \( \geq 5 \) ng/mL as a confirmatory test, the positive predictive value of an expert over reading the ECG to diagnose STEMI in the ICU was 33.3% (six of 18), whereas the negative predictive value was 96.4% (27 of 28).

Transthoracic echocardiograms were performed in six of the seven patients with troponin \( \geq 5 \) ng/mL and of those, four echocardiograms revealed global and regional wall motion abnormalities. In contrast, only four of 23 echocardiograms of patients with an ECG diagnosis of STEMI but troponin \(<5\) ng/mL showed global or regional wall motion abnormalities. Three of these patients had borderline troponin elevations.

There was a wide spectrum of underlying clinical diagnoses of patients with the ECG indication of STEMI, including acute cerebrovascular events or head trauma in 15 patients; respiratory failure resulting from pneumonia, acute respiratory distress syndrome, hemotherax, pneumoecomy and status asthmaticus in nine patients; and acute abdomen pre- and postoperatively in seven patients. Additional diagnoses included acute renal failure, hyperkalemia, hypertensive emergency with pulmonary edema, sepsis, pericarditis, infective endocarditis, and artifact. We did not systematically analyze the clinical context of the individual cases to try to determine the exact cause of the ECG abnormality. In a few cases, however, there was a likely cause established. These included artifact, hyperkalemia, pericardial irritation by a chest tube, pneumomediastinum, and pancreatitis. The most common cause of electrocardiographic STEMI was an acute intracranial event. Figures 2–4 are representative examples of ECGs in which the ECG was interpreted as STEMI both by computer interpretation and by expert opinion; the troponin elevation, however, was unremarkable.

**DISCUSSION**

Our study demonstrates that the ECG interpretation software diagnosis of STEMI is a common finding in the ICU. In our ICUs, approximately 3% of all ECGs display this phenomenon, and the average occurrence of electrocardiographic STEMI is approximately one case every week. Of note, in almost 40% of the cases even an ECG expert agreed with the ECG diagnosis. In the vast majority of cases, however, the patients most certainly did not have acute myocardial infarction. The main finding of this study, therefore, is that in ICUs, the ECG has a poor predictive value for acute myocardial infarction and lacks the specificity compared with non-ICU patients.

In patients who present with chest pain, the ECG is the primary decision or branch point in the acute coronary syndrome algorithm to determine the need for immediate reperfusion therapy (1–4). In this scenario, the ECG is associated with 34% sensitivity and 96% specificity for changes indicating myocardial infarction when expert evaluation is used (1). The GE-Marquette 12SL interpretation software has a similar reported sensitivity and specificity of 40% and 99%, respectively, for the diagnosis of acute myocardial infarction with a positive predictive value of 80% (17). In sharp contrast to these data, the most striking finding in our study was the extremely poor positive predictive value of the ECG (33.3%) to predict the possible diagnosis of STEMI in the ICU. The ECG, therefore, which is the gold standard for diagnosing clinical STEMI in patients who present with chest pain, may be a poor diagnostic modality to indicate STEMI in critically ill patients in ICUs. Under these circumstances, a bedside transthoracic or transesophageal echocardiogram may help increase or decrease the likelihood of STEMI (18).

In the ICU, important elements of the clinical history, specifically that of chest...
pain, are often lost because of a variety of ICU-specific factors such as shock, altered mental status, and mechanical ventilation. ECGs are often obtained for reasons other than chest pain such as arrhythmias, hypotension, agitation, pulmonary edema, or a sudden change in the vital signs. Under these circumstances if the ECG indicates STEMI, the question becomes how to interpret this ECG diagnosis and how to proceed with further diagnostic testing and treatment. Our study suggests that in the majority of cases, a cause other than myocardial infarction should be searched for, and emergent cardiac catheterization should probably be reserved for a very small number of cases in which the clinical picture or another imaging study such as the echocardiogram independently indicates a high likelihood of STEMI. Of note, only one of our patients with the ECG diagnosis of STEMI underwent emergent cardiac catheterization. This suggests that the treating physicians did not have a high degree of clinical suspicion for acute myocardial infarction.

The ECG diagnosis of STEMI in the ICU is reminiscent of the problem with troponin elevations in critically ill patients. Recent consensus statements from various cardiology societies recommend the use of ECG criteria and a rise and fall in cardiac serum markers as evidence of acute myocardial infarction (2–4). A number of studies, however, have shown
that acutely ill patients have conditions other than myocardial infarction that can cause elevations in serum troponin levels (19–22). Interestingly, even in the absence of an acute coronary syndrome, elevated serum troponin levels are a risk factor for mortality in ICU patients with critical illnesses (20, 21, 23). The exact relationship between troponin elevations and decreased survival is speculative but may be the result of subendocardial ischemia from a stress-induced increase in myocardial oxygen demand and/or diminished oxygen supply associated with cardiopulmonary compromise. It is of interest that the majority of our patients with an ECG diagnosis of STEMI did have some troponin elevation, albeit not to levels consistent with STEMI. Future larger studies comparing outcomes of critically ill patients with or without the ECG diagnosis of STEMI may shed light on whether this ECG sign by itself, even in the absence of a true acute myocardial infarction, has any prognostic significance.

There are multiple limitations to this study. First, not all patients with STEMI ECGs had concomitant serum troponin measurements necessitating the withdrawal of seven patients from the final analysis. Second, a troponin elevation of <5 ng/dL to indicate the probable absence of a true STEMI was chosen somewhat arbitrarily. Based on troponin data of patients with STEMI from our and other institutions, however, we felt that a peak troponin of <5 ng/mL reliably excluded STEMI (16). Third, the timing of troponin testing was not standardized nor were the reasons for the ECG acquisition. Fourth, because of the type and severity of the critical noncardiac illness, the majority of patients did not undergo coronary angiography. Finally, like with all retrospective studies, we were limited by the ability to gather all relevant clinical data in a systematic fashion.

CONCLUSIONS

STEMI in the ICU is a relatively common ECG reading both by standard interpretation software and by expert evaluation. In contrast to non-ICU patients who present with chest pain, the ECG STEMI diagnosis seems to be a nonspecific finding in the ICU that is frequently the result of a variety of nonischemic processes. The vast majority of such patients do not have frank STEMI. Participants in this CME activity should be better able able to evaluate the value of ECG interpretations and troponin measurements in the diagnosis of STEMI for ICU patients. Participants also should be better able to assess the need for emergency cardiac catheterization in this population.

ACKNOWLEDGMENTS

We acknowledge J. Warren Holshouser, MD, FACC, for his time and effort in interpreting the electrocardiograms, and Anne Focht, RN, MSN, for her assistance in data collection and analysis.

REFERENCES

9. Rosenbloom AJ: Massive ST-segment eleva-

Figure 4. Electrocardiogram of a 72-yr-old man with a history of diabetes, hypertension, chronic obstructive pulmonary disease, and end-stage renal disease who was admitted to the intensive care unit for hypertensive urgency with flash pulmonary edema. The interpretation software and expert evaluation both indicated ST-segment elevation myocardial infarction. The serum troponin peaked at 0.1 ng/dL and the echocardiogram showed an ejection fraction of 20–25% without regional wall motion abnormality. Before discharge, an adenosine nuclear perfusion study showed no perfusion abnormality.
tion without myocardial injury in a patient
with fulminant hepatic failure and cerebral
edema. *Chest* 1991; 100:870–872

10. Sampson M, Rose CE Jr: Reversible ST-
segment elevation associated with atelecta-
98:949–951

11. Slay RD, Slay LE, Luehrs JG: Transient ST
elevation associated with tension pneumo-
thorax. *JACEP* 1979; 8:16–18

12. Khairy P, Marsolais P: Pancreatitis with elec-
trocardiographic changes mimicking acute
myocardial infarction. *Can J Gastroenterol*
2001; 15:522–526

al: Pneumomediastinum mimicking acute
ST-segment elevation myocardial infarction.
*Int J Cardiol* 2007; 117:e73–e75

14. Littmann L, Taylor L 3rd, Brearley WD Jr:
ST-segment elevation: A common finding in
severe hypercalcemia. *J Electrocardiol* 2007;
40:60–62

Incidence, clinical findings, and outcome of
women with left ventricular apical balloon-
ing syndrome. *Am J Cardiol* 2007; 99:
182–185

troponin I in acute coronary ischemic syn-
dromes: Epidemiological and clinical corre-
lates. *Int J Cardiol* 2001; 77:215–222

17. GE Healthcare. Marquette™ 12SL™ ECG
Analysis Program: Statement of validation
and accuracy. 416791-003, Revision B, Feb-
ruary 1, 2007

18. Poelaert J: Use of ultrasound in the ICU. *Best
Pract Res Clin Anaesthesiol* 2009; 23:
249–261

troponin level is not synonymous with myo-
cardial infarction. *Int J Cardiol* 2006; 111:
442–449

ponin as a risk factor for mortality in critically
ill patients without acute coronary syndromes.

dial necrosis in ICU patients with acute non-
cardiac disease: A prospective study. *Intensive
Care Med* 2000; 26:31–37

and transient ST segment elevation during
bacterial shock in seven patients without ap-

implications of normal (<0.10 ng/ml) and
borderline (0.10 to 1.49 ng/ml) troponin el-
evation levels in critically ill patients without
acute coronary syndrome. *Am J Cardiol*
2008; 102:509–512