Interpretation of the electrocardiogram: clinical correlation suggested

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This editorial refers to ‘Local hospital vs. core-laboratory interpretation of the admission electrocardiogram in acute coronary syndromes: increased mortality in patients with unrecognized ST-elevation myocardial infarction’ by R. Vijayaraghavan et al., on page 31

Vijayaraghavan et al. have investigated the interpretation of the initial 12-lead electrocardiogram (ECG) in acute coronary syndrome (ACS) patients, focusing on the patient with potential ST segment elevation myocardial infarction (STEMI). The authors emphasize the importance of accurate ECG interpretation by the acute care physician (i.e. emergency physician, internist, and cardiologist).

The authors compared the initial interpretation of the initial admission ECG by the treating physician with the interpretation of the same tracing by a physician at the core electrocardiographic laboratory. The ECG interpreters at the site were acute care physicians (emergency physician, internist, and cardiologist); at the core lab, the ECG was interpreted by non-cardiologist physicians using specific definitions of abnormality—in this case, ST segment elevation was defined as >0.1 mV in two contiguous leads. The subgroup of patients used in the study were taken from the Canadian ACS Registry and comprised 1310 patients, of which 1202 had complete data and were used for analysis. The definition of STEMI included ST segment elevation and a positive biomarker. What is unclear is whether a positive biomarker was a single spot of STEMI included ST segment elevation and a positive biomarker.

Overall concordance between core-lab and site interpretation of the admission ECG was 62%. The calculated kappa value was 0.49, indicating modest agreement. When compared with the agreement group of the study, patients in the discordant subgroup were older with higher rates of diabetes mellitus, angina, myocardial infarction, congestive heart failure, coronary artery bypass grafting, and percutaneous coronary intervention. Patients with core-lab-defined ST segment elevation that was not noted by the treating site, who also had a positive biomarker, were significantly less likely to receive aspirin, heparin, and reperfusion therapy when compared with patients with core lab-site ECG interpretation agreement. After adjusting for other validated prognostic factors, site-unrecognized ST elevation was independently associated with higher mortality, most probably because of the increased rate of co-morbidities in this subgroup.

It is interesting to note that a minority of this ‘site-unrecognized STEMI’ group did, in fact, receive reperfusion therapy somewhat later in the course of care (53 patients or 14% of this subgroup). Furthermore, another small portion of these patients had significant contraindication to fibrinolysis and, therefore, did not receive reperfusion therapy (92 patients or 24% of this subgroup).

The authors concluded that significant rates of disagreement are found in the recorded interpretation of patients presenting with ACS—in particular, rates of ST segment elevation determination. Further, they note that the unrecognized STEMI is associated with underutilization of evidence-based therapies as well as worsened 1-year outcome (i.e. increased mortality).

The study of Vijayaraghavan et al.1 is quite interesting for a number of reasons; it focuses on the all-important ability of the physician to interpret the ECG in the acute care setting—we must not lose sight of this message. Yet, there are several issues of concern that must be discussed, including the definition of acute myocardial infarction, the presence of non-STEMI ST segment elevation syndromes, outcome issues in the interpretation disagreement group, and reality of acute care clinical practice. Much of the disagreement between interpretations of identical ECGs may be due to differences in the clinical context in which the ECG is used. One interpretation takes place at the point of care, whereas the other takes place in isolation without the benefit of having a patient to assess. Point-of-care interpretation probably benefits from the patient’s clinical status (are they pain free?) and availability of previous tracings. It should be noted that the disagreement between ECG interpretations worked both ways; there were 335 cases where the site noted ST-elevation that was ‘missed’ by the core-lab interpretation, compared with...
715 cases of ST-elevation detected by the core lab and ‘missed’ by the site. Because there was no final adjudication, to claim that one site vs. the other rendered the ‘correct’ interpretation seems far fetched. In addition, the vast majority of discordant interpretations were classified as ‘other’, making an assessment of the clinical impact problematic.

The definition of STEMI in the study of Vijayaraghavan et al. includes the presence of ST segment elevation and a positive biomarker indicative of myocardial injury. Importantly, it is unclear if the positive biomarker is based upon a single test or a series of tests with the ‘typical rise and fall’ pattern. In fact, the most appropriate definition of acute myocardial infarction, jointly developed by the European Society for Cardiology and American College of Cardiology, requires a typical rise and fall of biomarker along with at least one of the following findings: ischaemic clinical symptoms, ECG changes indicative of ischaemia (ST segment deviation), the development of pathological Q waves on the ECG, or coronary artery abnormality based upon intervention data. Clearly, the use of the single positive troponin value criterion introduces the possibility of patient inclusion in the STEMI group when myocardial infarction has not occurred. Consider two possible scenarios which would be labelled STEMI in this type of analysis and yet do not represent ST segment elevation myocardial infarction: (i) the patient with past myocardial infarction complicated by left ventricular aneurysm with resultant, persistent ST segment elevation; and (ii) the patient with a left ventricular hypertrophy electrocardiographic strain pattern. In these two scenarios, ST segment elevation meeting the authors’ criteria for fibrinolysis would be present; further, a single, minimally positive troponin in this setting without the abnormal pattern suggestive of infarction does not support the diagnosis of myocardial infarction—thus the term and diagnosis ‘STEMI’ could be misapplied.

The patient with chest pain and electrocardiographic ST segment elevation presents a challenge to the treating physician. He or she is charged with a rapid interpretation of the entire clinical picture, including the ECG, and a determination of the need for urgent reperfusion therapy—at times with limited data while the demands of other patient care continues. As has been reported, all patients with electrocardiographic ST segment elevation and chest pain are not experiencing STEMI. In fact, only a minority of these patients are suffering from STEMI. In the study of Vijayaraghavan et al., it is unclear if all patients with ST segment elevation and positive biomarkers were experiencing ACS in general or STEMI in particular. Consider the patient with the benign early repolarization pattern and minimally positive troponin value who presents with an emergent hypertensive state—again a patient with ST segment elevation and a positive biomarker who is not experiencing STEMI.

Unfortunately, ST segment elevation, by itself, is a less than sensitive indicator of STEMI. In fact, numerous clinical and/or electrocardiographic entities present with ST segment elevation. ST segment elevation is encountered in approximately one-fifth of all ED chest pain patients—the majority of these patients will ultimately be diagnosed with a non-ACS cause of the ST segment abnormality; STEMI accounts for only 20% of these patients with electrocardiographic ST segment elevation. The electrocardiographic differential diagnosis of ST segment elevation in the adult chest pain patient is extensive, ranging from STEMI, left ventricular aneurysm, myocarditis, and cardiomyopathy to bundle branch block, pericarditis, benign early repolarization, left ventricular hypertrophy, and ventricular paced rhythm. The most common causes of ST segment elevation among chest pain patients are non-infarction diagnoses—namely bundle branch block and left ventricular hypertrophy patterns. This observation is correct regardless of the chest pain population considered, including the pre-hospital, Emergency Department (ED), and Coronary Care Unit. In these populations, non-STEMI electrocardiographic patterns are the most frequently encountered ST segment elevation entities among adult patients with chest discomfort.

Interestingly, patients in the ECG interpretation disagreement group, the presumed missed STEMI population, had a higher rate of all-cause mortality with a markedly lower rate of anti-coagulant, antiplatelet, and fibrinolytic therapies. Certainly, a number of these patients were probably experiencing STEMI and yet did not receive immediate reperfusion therapy. This subgroup was composed of a generally older, more chronically ill patient population. Thus, for the reasons noted above, a portion of these patients were not experiencing STEMI and therefore did not receive ACS therapy. Furthermore, this older patient group with extensive past cardiovascular history had a higher rate of all-cause mortality during the follow-up period. Again, undoubtedly, a segment of this patient subgroup were probably experiencing STEMI but did not receive therapy early in the hospital course, whether due to diagnostic errors or to complicated medical presentations.

Regardless of the results of this study, we believe that the physician reviewing the ECG in the suspected ACS patient must be an expert in electrocardiographic interpretation. Emergency physicians are such experts. In the often hectic ED environment with multiple, near simultaneous demands on his/her attention, the emergency physician interprets the ECG rapidly and accurately, often with minimal clinical data and without benefit of prior ECGs for comparison. Importantly, this review of the ECG occurs during active patient care such that the interpretation is applied in real time to the management plan—not after the fact, long after patient care decisions have been made and carried out. Emergency physicians are experts in the interpretation of the real-time ECG.

The medical literature supports this contention from numerous perspectives. General electrocardiographic interpretation abilities are quite impressive, with consistently high rates of agreement noted between the emergency physician and cardiologist. For instance, Zappa et al. reported a low rate of disagreement between senior emergency medicine residents and staff cardiologists; no clinically significant discrepancies were noted when the ECGs which were the subject of disagreement were reviewed. Schaffer and colleagues considered the electrocardiographic interpretation of attending physicians in emergency medicine and cardiology, reporting that a very high rate of agreement was found—96% of these interpretations were agreed upon by these two physician groups. Additional reviews of the topic demonstrate similar findings—high rates of agreement in interpretation between cardiologists and emergency physicians.

If one focuses on the ACS electrocardiogram, very high rates of correct interpretation are found; again it must be stressed that this review occurs in real time with minimal clinical data available to
assist with the ultimate ECG diagnosis.\textsuperscript{12–14} Aufderheide and colleagues\textsuperscript{12} reported a prospective study which involved pre-hospital chest pain patients undergoing a pre-hospital 12-lead ECG, demonstrating that the prospective interpretations of practise emergency physicians were similar to those of a group of cardiologists retrospectively in the electrocardiographic diagnosis of STEMI. A survey of practising emergency physicians evaluated the ability to determine the aetiology of electrocardiographic ST segment elevation in a hypothetical chest pain patient—and make treatment recommendations based upon this interpretation.\textsuperscript{13} Approximately 95\% of the interpretations were performed correctly with minimal clinical information available at the time of interpretation. A real-time review of the electrocardiographic interpretations of ED chest pain patients with ST segment elevation revealed a very high rate of correct interpretation with an error rate of only 6\%; misinterpretations were not clinically significant in any instance.\textsuperscript{14}

This study of Vijayaraghavan et al.\textsuperscript{1} explores the always important topic of ECG interpretation in the presumed STEMI patient.\textsuperscript{1} We commend the authors for their efforts in this area and for publishing such excellent data from one of the largest ECG registries (GRACE) to date.\textsuperscript{15} Clearly, the ECG must be interpreted correctly in the ED such that appropriate therapy can be offered. In the STEMI patient, urgent reperfusion therapy decisions must be made within minutes of arrival in the ED, largely resting on the interpretation of the 12-lead ECG. Because of this need for rapid decision making, the emergency physician must be an expert in the interpretation of the 12-lead ECG. The emergency physician must also be an expert in the medical decision making that occurs in the context of clinical care. The ECG is but a portion of the data used in this decision making, and clinical correlation is the key to optimal management.

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**References**


