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The effect of changing diagnostic algorithms on acute myocardial infarction rates

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Abstract

Purpose—Population rates of acute myocardial infarction (AMI) are changing. Consistent case definitions to evaluate these trends and make comparisons are essential. The World Health Organization (WHO) AMI diagnostic algorithm and clinical judgments were the standards for classification. However, in recent years, five new algorithms, to include diagnostic advances, are advocated by professional organizations. This study compares AMI rates derived from six algorithms and the impact of troponins on those rates.

Methods—The authors utilize the population-based Minnesota Heart Survey hospital data in 1995 and 2001 to compare six published diagnostic algorithms and the impact of troponins.

Results—In 1995 differences in AMI rates between algorithms ranged from 281/100,000 to 440/100,000 for men and 98/100,000 to 139/100,000 for women. The use of troponin, a more sensitive biomarker, adds to the differences by increasing eligible cases. Using 2001 data in patients where creatine kinase and troponin were simultaneously measured, a 64% and 95% increase in AMI rates among men and women, respectively, was observed.

Conclusions—Accurate and consistent AMI definitions are crucial for clinical trials, epidemiology and public health research. Demonstrated here is the sensitivity of AMI rates to changing case definitions and the biomarker troponin.

Keywords

Acute myocardial infarction(AMI); AMI rates; AMI algorithms; registries; creatine kinase; troponins

Introduction

Coronary heart disease (CHD), commonly manifest as acute myocardial infarction (AMI), is a major source of morbidity and mortality worldwide (1). Assessment of the incidence, prevalence and trends in AMI is critical to understanding this epidemic and explicit case definitions are essential methodologic tools. Historically, the World Health Organization (WHO) criteria form the basis for the diagnosis of an AMI case (2). Developed in the 1970s for European AMI registries, the WHO definition emphasizes classical symptoms, elevation

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in cardiac biomarkers and electrocardiographic (ECG) changes. However, the WHO criteria are ambiguous, lacking precise definitions and are frequently misinterpreted (3). As a result of this ambiguity and advancing diagnostic technology, cardiovascular disease experts in the 1980's developed new criteria for AMI definitions with algorithms to better identify cases. These new criteria included efforts in the United States and Europe (4, 5).

Technological innovation in enzymes and other biomarkers in the late 1990s led to new demands for modification of the criteria. It was also apparent that the nature of the disease was changing and the classical Q-wave AMI was becoming less common with milder and more varied presentations emerging. The European Society of Cardiology (ESC) and American College of Cardiology (ACC) developed a new criteria set for clinical and research studies in 2000 (3). The new algorithm recognized modern biomarkers and the potential for imaging studies in the clinical and research settings. These criteria represented an advance but were developed for clinicians who needed to make the diagnosis in the acute clinical setting as opposed to retrospective studies where complete data are available (6-8). A 2003 workshop by the American Heart Association (AHA), World Heart Federation (WHF) and WHO developed criteria specifically for epidemiology studies and classification methods for developing countries where advanced diagnostic measures might not be available (9). In 2007, the ESC, ACC, AHA and WHF proposed a new “universal definition of myocardial infarction” based on diagnostic advances in cardiology and concerns regarding the 2000 recommendations (10).

The current study examines the published criteria for diagnosing AMI using Minnesota Heart Survey (MHS) population-based data in 1995 and 2001 (11). We hypothesize rates for prevalence, incidence and trends will differ significantly based on the diagnostic algorithm selected. In addition to evaluating the rates produced by the different algorithms, data are presented to quantify the impact of the newest biomarker, troponins, on AMI diagnosis.

Methods

Diagnostic algorithms for AMI include three basic elements: symptoms, biomarkers and ECGs. The most advanced algorithms also include imaging techniques, such as computerized tomography and magnetic resonance imaging, but these are still rarely used in either clinical or epidemiologic applications (3,10).

Six current algorithms are compared including: 1. MHS (11); 2. AHA/WHF/WHO (9); 3. ESC/ACC (3); 4. WHO (2); 5. Monitoring of Trends and Determinants of Cardiovascular Disease (MONICA) (5) and; 6. ESC/ACC/AHA/WHF (12). In Table 1, the different elements and criteria for an AMI case for the six algorithms are described in detail. Signs and symptoms are generally consistent across algorithms, but not identical. Biomarkers reflect the evolution from lactic acid dehydrogenase (LDH) and serum glutamic oxaloacetic transaminase (SGOT) to troponin with the most recent criteria eliminating the earlier markers such as LDH, SGOT, myoglobin and total creatine kinase (CK). ECGs are also de-emphasized in recent algorithms. This parallels the reduced prevalence of classical ST elevation and Q-wave AMIs as many biomarker increases are associated with non-specific ECG changes. Most algorithms also include autopsy findings. Patients who die without autopsy, biomarkers, ECGs or other evidence are not included. Many algorithms downgrade an AMI diagnosis after a rise in biomarkers with a surgical procedure or trauma (See Table 2 footnote). Although some algorithms use the classifications probable and possible, this study compares those with the definite AMI only.

Study population

The six algorithms for AMI classification were tested on the 1995 MHS population survey (4). This community AMI surveillance samples patients aged 30-74 hospitalized with suspected AMI in the Minneapolis/St. Paul metropolitan area. In the 1995 annual survey, troponins were rarely used by the hospitals and CK or creatine kinase myocardial band (CKMB) was the standard. In the 2001 survey, troponins were the principal biomarker, though frequently used in combination with CK. The 2001 data were used to test the effect of troponins on rates using the AHA algorithm (9).

The MHS draws from the seven county metropolitan area of Minneapolis/St. Paul in Minnesota (population 2.6 million: 2000 census). The hospitalized population is principally white (>90%). The Minnesota Hospital Association provided the medical record numbers of all patients 30-74 years old who were discharged from metropolitan area hospitals with a discharge diagnosis of acute CHD in 1995 and 2001. The target International Classification of Diseases (ICD)-9 codes were 410 (AMI) and 411 (unstable angina). We eliminated those with a target code of ICD-9 410.x2 which identified uncomplicated rehospitalizations for follow-up treatment for an AMI that occurred within the previous six weeks. We excluded non-acute hospital admissions, in-hospital MI, angina and transfers (in or out of hospital). In 1995, 22 of 23 hospitals participated and in 2001, 20 of 21 were included. The hospital omitted in both surveys was a small rural hospital accounting for <1% of CHD discharges.

The sampling fraction varied by surveillance time frame. In 1995, a randomly selected 40% sample of men and 80% sample of women with ICD-9 410 and 411 were reviewed. In 2001 we randomly sampled 42.5% of men and 85% of women from the metropolitan hospitals and 50% of men and 100% of women from smaller community hospitals (<100 admissions) over six months (July to December). To enhance the efficiency of AMI case finding, the ICD-9 411 discharges, in which a biomarker was elevated above the hospital-specific upper limit of normal, were completely abstracted.

The medical records of the selected hospitalizations were reviewed by trained nurses with a written protocol. Information was obtained on signs and symptoms, medical history, cardiac biomarker levels, clinical complications, therapy, medical procedures and, when applicable, autopsy results. Up to four ECGs were photocopied and coded according to the Minnesota Code (9). Using the six diagnostic algorithms in Table 2, each case was declared a definite, probable, possible AMI, or not an AMI.

To evaluate the differences associated with the advent of troponins, we utilized the 2001 cohort. In this group, because of clinician questions about a new technology, many of the hospitals simultaneously measured troponins and CK or CKMB. Among those cases with both biomarker measures, we ascertained the proportions of AMI with one or both biomarkers positive.

The study protocol was approved by the University of Minnesota Institutional Review Board and the review boards of participating hospitals.

Statistical analysis

The primary analysis is descriptive in nature. Each AMI algorithm was applied to the MHS cases to determine the number of validated definite AMIs for a particular definition. The observed numbers of cases were multiplied by the inverse of the sampling fractions for each gender to estimate the number of AMIs in the metropolitan area in 1995 and in 2001. AMI rates are calculated for each algorithm by dividing the number of AMIs by the population of individuals aged 30-74 in the Twin Cities estimated from the U.S. Census for 1995 and 2001.

In order to compare data in the troponin era with data from the CK era, dual-measured patients were used to develop a correction factor. Patients who were CK or CKMB positive and troponin negative plus those with both biomarkers positive were summed and divided by the sum of all patients with any positive test to produce CK adjusted ratios. The AHA criteria, designed for epidemiology studies, were used for the AMI definition (9). Of the 440 people with abnormal CK levels aged 30-74 with a definite MI defined by AHA criteria in 2001, 18 were missing troponin data and five had a normal troponin level.

Results

The population samples used to test the algorithms included 3197 cases in 1995 and 1617 cases in 2001. CK biomarkers were reported for all of the 1995 cases. In 2001, 748 (46%) patients had both troponin and CK measured.

Table 2 shows the 1995 hospitalization rates for definite AMI per 100,000 population using the six different published algorithms with and without the downgrade for muscle cutting procedures or trauma. The rates for men vary widely from 440/100,000 for the WHO standard to 253/100,000 for the MONICA algorithm with the downgrade, a difference of 58%. The other methods range between those two extremes. Among women, the WHO and ESC/ACC algorithms result in 139 cases/100,000 population and the MONICA algorithm with the downgrade results in 92/100,000 population, a difference of 51%. Table 2 shows the number of cases of ICD 411 (unstable angina) that were adjudicated.

The results of switching to the more sensitive troponin assays are demonstrated in the 2001 data (Table 3). The measurement of troponins adds a considerable number of cases to the definite AMI category. The CK adjusted ratios were 0.61 for men and 0.51 for women ($t=2.67$, $p=.008$). As shown in Table 4, the attack rate using the AHA algorithm with any positive biomarker was 417/100,000 and 152/100,000 for men and women, respectively. After adjustment of troponin to CK, the rates are 254/100,000 and 78/100,000 for men and women, respectively. Compared to the 1995 data, it is apparent that the adjusted attack rate demonstrates a fall in definite AMI over time, whereas using just troponin levels would demonstrate an increase.

Discussion

The accurate and rapid diagnosis of AMI has important therapeutic implications for clinicians, but consistent case definitions are critical for epidemiologists who seek to describe the epidemic and evaluate trends in incidence and prevalence (13). Research scientists must select from the various definitions to ensure consistent endpoints in clinical trials where previous AMI is used to select the target population, or, is an endpoint of the study. This study shows the dramatic effect of applying various algorithms on definite AMI rates.

The definition of AMI is evolving. The historic WHO criteria (2) were liberally interpreted by clinicians, trialists and researchers. This definition failed to account for changing biomarkers and increasing numbers of patients without classical ECG findings. A particular challenge was posed by myocardial specific CKMB and, more recently, the widespread use of troponin. The latter is highly sensitive and specific for myocardial injury, able to detect much smaller areas of myocardial damage (14,15,16).

The need to describe cases in the setting of changes in disease presentation and biomarkers resulted in the proposal of a number of new diagnostic algorithms in recent years. These algorithms are designed to have utility for clinicians, trialists and epidemiologists. Beginning with the basic WHO criteria of symptoms, biomarkers and ECGs, each new

algorithm developed a unique set of criteria for AMI diagnosis (2-5,9,12). Unfortunately, for consistency of epidemiologic statements, these algorithms chose different criteria and so, not surprisingly, yield different results.

In this study we compare published algorithms to calculate AMI rates from a population survey of hospitalized AMI in a large metropolitan area. These comparisons reveal vastly different rates. It is apparent that any attempt to make comparisons or evaluate trends must recognize these differences and utilize a consistent method or adjustment factors.

The advent of troponins further complicates the issue, particularly in attempting to evaluate trends. In this study, we were fortunate to have a large portion of the population with simultaneous CK and troponin measurements. Adjustment factors based on this overlap population allow examination of the differences. The magnitude of difference observed here and by others provides an alert for those attempting to compare rates in the pre- and post-troponin eras.

Others have also observed differences in attack rates, incidence and prevalence in population-based and clinical trial studies with different algorithms and biomarkers. In Finland, the application of the AHA algorithm resulted in an 86% increase in AMI attack rate compared to the rate by the MONICA algorithm (17). A study by Roger, et al. in Rochester, Minnesota, found a 74% increase in estimated AMI attack rates associated with use of troponin measures (18). A review of published studies comparing AMI diagnosis with and without troponin, found differences compared to total CK from 0 to 320% with troponin and 3.9 to 195% with CKMB (19). With one exception (20), the advent of the more sensitive biomarker has resulted in higher rates of AMI diagnosis. In this study and another, physicians were discovered to be reluctant to label mild elevations in troponin as AMI (18,20). A data specified diagnostic algorithm does not directly include medical judgment.

While troponins do diagnose smaller AMIs, it is well recognized they are a predictor of mortal outcomes with or without companion elevations in CK (21-23). The TACTICS-TIMI 18 trial evaluated 895 patients with suspected acute coronary syndromes with troponins and coronary angiography. Those patients with positive troponins without angiographic coronary artery disease had a six month rate of death or reinfarction of 3.1% while those with negative troponins and angiography had an event rate of 0% (23). In addition, a troponin positive AMI with a negative CK has similar mortal outcomes as CK positive AMI after adjustment for age, gender, the history of cardiovascular disease and cardiac procedures (24). Assuming that absence of a troponin measure in 1995 resulted in an underestimate in what we now might call AMI, we would speculate that the underlying rate of AMI (or at least of more severe AMI) did decrease, and that milder AMIs were identified with the advent of troponins.

As with previous biomarkers of AMI, there is a growing knowledge about false-positive troponin results. These now include congestive heart failure, pulmonary embolism, rhabdomyolysis, chronic renal failure, myocarditis, burns, extreme exertion, coronary spasm, arrhythmias, pericarditis, septicemia, trauma and chemotherapy (25,26). This list of factors which elevate troponin levels are thought to represent smaller amounts of myocardial damage. False-positive tests for AMI will undoubtedly increase in number as the test is more widely applied or more sensitive measures are developed. This knowledge is important in evaluating AMI algorithms as those which require rising and/or falling biomarker levels are likely to be more accurate than the acceptance of one elevated level.

There are several limitations to this study. The algorithms described here (Table 1) are the result of the interpretation of published literature. Some algorithm descriptions are more detailed than others. The trend has been toward more explicit rather than less explicit

definitions in algorithms, but judgments are involved in the setting of limited specificity. A particular point of question is the implication of missing information for the implementation of each algorithm as some are explicit about missing values and others are not. This study, while population-based, is also dependent on the availability of accurate information from many hospitals and on medical judgment to perform the tests needed for the present research. Records in the current environment are quite complete but the availability of additional laboratory testing or multiple ECGs is not perfect. This is a particular problem when the patient expires shortly after admission. These cases may have limited data and expert judgment plays a role in their classification. Algorithms used by epidemiologists are likely to under-diagnose such cases. Furthermore, silent AMI and AMI leading to sudden death out of hospital are excluded in any hospital-based study.

In our comparison of patients with both biomarkers (CK and troponin), it is possible that selection bias was introduced by clinicians who ordered both tests for a subset of patients. However, the proportion of patients with both measures was substantial (46%) and the practice was not limited to certain hospitals. It is likely that this was the testing habit of some clinicians or they sought to resolve ambiguous patients. Currently, many hospitals offer CK only by special request.

Finally, troponin assays are marketed by multiple manufacturers, often using different reagents (27,28). We accepted individual hospital standards for normal without adjustment for manufacturer. The addition of new and ultra-sensitive troponin measures, now in the fourth generation, will add to the complexity of interpreting any results and likely increase the rates of AMI diagnosis (29,30). The more sensitive measures are a challenge for clinicians who rely more on ‘clinical judgement’ for the diagnosis in the setting of small troponin elevations. For epidemiologists, using data-based algorithms which exclude clinical judgement, there are also challenges in interpreting ultra sensitive troponin data and further increasing AMI rates by including potential false-positives. The advent of other new technologies using echocardiography, magnetic resonance imaging, radionuclide approaches and computerized tomography also add to the diversity of testing and the challenges in defining events (25). These and other as yet unforeseen changes, pose future challenges for case definition for clinicians, trialists, and epidemiologists. For the latter, the use of consistent methods allow the estimates of trends in disease rates, comparative studies between populations and estimates of the burden in the population.

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List of abbreviations and acronyms

AMI	Acute myocardial infarction
WHO	World Health Organization
CHD	Coronary heart disease
ESC	European Society of Cardiology
ACC	American College of Cardiology
AHA	American Heart Association
WHF	World Heart Federation
MHS	Minnesota Heart Survey
ECG	Electrocardiogram
LDH	Lactic acid dehydrogenase
SGOT	Serum glutamic oxaloacetic transaminase
CK	Creatine kinase
CKMB	Creatine kinase myocardial band
MONICA	Multinational MONItoring of trends and determinants in CArdiovascular disease

Table 1

Diagnostic Elements in AMI Algorithms

	Algorithms					
	WHO	**MHS**	**MONICA**	**ESC/ACC**	**AHA**	**ESC/ACC/AHA/WHF**
Publication year	1976	1987	1999	2000	2003	2007
	Emphasis Level*					
ECG	3	3	3	3	3	1
Biomarkers	2	2	2	3	3	3
Symptoms	2	2	2	3	2	2
Autopsy	2	3	3	3	3	2
Iatrogenic AMI	0	2	0	0	0	3
Delayed death (30 days)	0	3	3	3	3	3

*Importance in algorithm

0=Not considered; 1=Rarely considered; 2=Usually considered; 3=Always considered

WHO=World Health Organization; MHS=Minnesota Heart Survey; MONICA=WHO Monitoring Trends and Determinants of Cardiovascular Disease; ESC=European Society of Cardiology; ACC=American College of Cardiology; AHA=American Heart Association; WHF=World Heart Federation

Table 2

Twin Cities Hospitalized Rates of Definite AMI per 100,000 in 1995

	Definite AMI 410/41 1 Rate		Definite AMI 410 Only		Definite AMI 411 Only Rate	
Algorithm	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
Discharge Diagnosis (any position)	878.4 (416.7 - 1340.0)	308.0 (108.8 - 507.3)	417.0 (207.5 - 626.5)	141.8 (51.4 - 232.1)	461.4 (206.2 - 716.5)	166.3 (56.8 - 275.8)
MHS (11)	385.9 (365.6 - 406.3)	125.4 (117.5 - 133.3)	330.7 (319.0 - 342.3)	115.1 (110.8 - 119.4)	55.3 (45.5 - 65.0)	10.3 (7.4 - 13.2)
AHA (9)	423.8 (403.1 - 444.5)	137.6 (129.6 - 145.6)	345.6 (334.5 - 356.7)	120.2 (116.2 - 124.2)	78.2 (66.8 - 89.5)	17.4 (13.7 - 21.0)
AHA with Downgrade (9)	358.8 (338.5 - 379.0)	121.0 (113.1 - 128.9)	318.9 (306.6 - 331.2)	112.5 (108.0 - 117.0)	39.9 (31.4 - 48.4)	8.5 (5.9 - 11.2)
ESC ACC (3)	432.0 (411.4 - 452.7)	138.5 (130.5 - 146.5)	343.2 (332.0 - 354.4)	121.0 (117.0 - 124.9)	88.8 (77.0 - 100.7)	17.5 (13.8 - 21.1)
WHO (2)	439.6 (418.9 - 460.3)	139.4 (131.4 - 147.4)	350.0 (339.1 - 360.9)	120.1 (116.1 - 124.1)	89.6 (77.7 - 101.5)	19.3 (15.6 - 23.1)
WHO with Downgrade (2)	346.6 (326.4 - 366.7)	116.9 (109.1 - 124.8)	317.5 (305.1 - 329.9)	111.5 (106.9 - 116.0)	29.1 (21.8 - 36.4)	5.5 (3.4 - 7.6)
MONICA (5)	281.1 (261.9 - 300.2)	97.8 (90.3 - 105.3)	263.7 (249.9 - 277.5)	95.8 (90.6 - 101.0)	17.4 (11.7 - 23.2)	2.0 (0.7 - 3.3)
MONICA with Downgrade (5)	253.3 (234.7 - 271.8)	92.2 (84.9 - 99.6)	246.2 (232.2 - 260.3)	91.1 (85.8 - 96.4)	7.0 (3.4 - 10.6)	1.2 (0.1 - 2.2)
ESC/ACC/AHA/WHF (12)	385.0 (364.6 - 405.4)	122.3 (114.4 - 130.2)	317.0 (304.7 - 329.3)	107.5 (102.8 - 112.3)	68.0 (57.3 - 78.7)	14.7 (11.4 - 18.1)
ESC/ACC/AHA/WHF with Downgrade (16)	332.1 (312.2 - 352.0)	109.8 (102.1 - 117.6)	294.8 (281.8 - 307.7)	102.0 (97.0 - 107.0)	37.3 (29.1 - 45.6)	7.9 (5.3 - 10.4)

Age-adjusted to 2000 U.S. population

Downgrade means applying the following to creatine phosphokinase (CPK) or creatine kinase M band (CKMB) in the given algorithm:

CPK Downgrade: Trauma, muscle cut procedure (4 days), CABG surgery (4 days)

CKMB Downgrade: Muscle cut procedure (4 days), CABG surgery (4 days)

WHO=World Health Organization; MHS=Minnesota Heart Survey; MONICA=WHO Monitoring Trends and Determinants of Cardiovascular Disease; ESC=European Society of Cardiology; ACC=American College of Cardiology; AHA=American Heart Association; WHF=World Heart Federation

Table 3

Agreement and disagreement of normal or abnormal myocardial infarction status according to creatine kinase (CK) and Troponin

MEN

		CK		
		Normal	**Abnormal**	**Total**
Troponin	Normal	0	2	2
	Abnormal	161	249	410
				412
Adjustment Factor (estimated proportion with abnormal myocardial status if only CK were used):	**0.61**			

WOMEN

		CK		
		Normal	Abnormal	Total
Troponin	Normal	1*	5	6
	Abnormal	164	166	330
				336
Adjustment Factor (estimated proportion with abnormal myocardial status if only CK were used):	**0.51**			

* Diagnosis by ECG only

Table 4

AMI per 100,000 Population*, Age 30-74 in 2001

	Definite 410/411 AMI Rate	
	Male	**Female**
Discharge diagnosis (any position)	671	261
Attack rate (Definite 410/411)**	417	152
Adjustment to CK***	254	78

* Age-adjusted to 2000 U.S. population

** AHA Algorithm with downgrade

*** Correction coefficient for men (0.61) and women (0.51)