CME Information: Troponin Testing and Coronary Syndrome in Geriatric Patients With Nonspecific Complaints: Are We Overtesting?

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CME

Troponin Testing and Coronary Syndrome in Geriatric Patients With Nonspecific Complaints: Are We Overtesting?

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ABSTRACT

Background: Elderly patients presenting to the emergency department (ED) with nonspecific complaints (NSCs) often undergo troponin testing to assess for atypical acute coronary syndrome (ACS). However, the rate of ACS and utility of troponin testing in this population is unknown. We sought to determine the rate of ACS and diagnostic yield of troponin testing in elderly patients with NSCs.

Methods: We retrospectively identified all patients aged ≥ 65 years triaged in the ED with NSCs from January 1, 2017, to June 30, 2017. NSCs were defined a priori and included complaints such as weakness, dizziness, or fatigue. NSCs were verified in ED provider notes by trained abstractors blind to testing results. Exclusions were focal chief complaint in provider notes, fever, and no troponin ordered. ACS was strictly defined and independently adjudicated by two trained physician researchers blind to the study hypothesis. We calculated the proportion of patients with ACS within 30 days and the test characteristics of troponin to diagnose ACS.

Results: Screening identified 1,146 encounters, and 552 were excluded for fever or focal chief complaints in the provider notes. Of the remaining 594 patients, troponin was ordered in 412 (69%), comprising the study cohort. The mean (±SD) age was 78.7 (±8.3) years, with 58% female and 75% admitted. Troponin elevation occurred in 81 patients (20%). ACS occurred in 5 of 412 (1.2%). Troponin was 100% sensitive (95% confidence interval [CI] = 48% to 100%) and 81% specific (95% CI = 77% to 85%) for ACS. Of patients with elevated troponin, 93.8% were false positives (no ACS). All patients with troponin elevation were admitted, but only one underwent angiography and no patients received reperfusion therapy.

Conclusions: While consideration for ACS is prudent in selected elderly patients with NSCs, ACS was rare and no patients received reperfusion therapy. Given the false-positive rate in our study, our results may not support routine troponin testing for ACS in this population.

Patients aged 65 years or older account for approximately 15% of all emergency department (ED) visits in the United States.[1] These elderly patients often require significant resource utilization and are at increased risk of adverse outcomes such as functional decline, prolonged hospitalization, and death.[1–5] The assessment of this high-risk population can be complicated by the fact that elderly patients frequently

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present with nonspecific complaints, such as "weakness," "dizziness," or "not feeling well."[6,7] Furthermore, the medical history can be obscured by comorbidities, polypharmacy, and cognitive or functional impairment.[6,8] Elderly patients in the ED are often diagnosed with serious or life threatening acute medical problems, including those who present with vague or nonspecific complaints.[9]

Since nonspecific complaints often have a broad differential diagnosis and there are no recommended diagnostic algorithms for patients with nonspecific presentations, practitioners often embark upon extensive testing to assess for a wide array of serious conditions.[10] Acute coronary syndrome (ACS) is among the life-threatening conditions in the differential diagnosis of elderly patients with nonspecific complaints. Compared to younger populations, the elderly with ACS more frequently present without chest pain[11] and up to 20% of elderly patients with myocardial infarction may present with weakness as part of the chief complaint.[12,13] Further, cardiovascular disease is the leading cause of mortality and morbidity in the elderly.[14,15] Despite this, the frequency of ACS among elderly ED patients with nonspecific complaints has not been previously defined. Assessment for the presence of ACS typically includes troponin testing, but the utility of routine troponin testing in elderly patients with nonspecific symptoms is also unknown. The primary objectives of this study were to determine the frequency of ACS in elderly patients presenting to the ED with nonspecific complaints and to define the frequency and utility of troponin testing in this population.

METHODS

Study Design and Setting

This is a retrospective study of patients seen at an academic Level I trauma and tertiary referral center in the United States. The hospital is located near the center of a major metropolitan area and the ED sees approximately 100,000 patients per year. This study was approved by the local institutional review board.

Patient Identification and Data Abstraction

The target population was patients aged ≥ 65 years presenting to the ED with nonspecific complaints who underwent troponin testing. The cutoff of 65 years is consistent with previous studies and definitions[16] and based on evidence that patients older than 65 have an increased mortality and higher rate of hospitalization due to cardiac vascular disease.[17,18] The electronic medical record (EMR) was searched for patients registered in the ED aged 65 or older with triage chief complaints representing vague or nonspecific presentations. Potential triage complaints were defined a priori, including weak or weakness, dizzy or dizziness, fatigue, lethargy, altered mental status, light-headed, medical problem, examination requested, failure to thrive, or "multiple complaints." These complaints were based on a combination of previous definitions of nonspecific presentations and institutional EMR codified complaints suggesting patient inability to articulate the specific reason for visit in triage.[8,19] The EMR search included the 6-month period from January 1, 2017, through June 30, 2017.

Since some patients receive a triage complaint different than the chief complaint documented by the provider, we then performed a review of the provider note for each encounter with a nonspecific triage complaint. If the provider documented a focal chief complaint (e.g., any focal pain or injury complaint, shortness of breath, vomiting, diaphoresis, syncope, fever, cough, focal neurologic deficit), then the patient was excluded. In cases of multiple complaints, unless otherwise specified, the first symptom mentioned in the provider's note was counted as the chief complaint. Secondary focal complaints were allowed if a nonspecific complaint was documented first or as primary.

Determination of a "nonspecific complaint" was made by two trained physician researchers who were blind to test results, including troponin. The ED physician note was opened in the EMR and a determination was made about whether the patient had presented with a nonspecific chief complaint. This determination was documented on a standardized data collection sheet. After a nonspecific chief complaint had been verified, the abstractor determined whether a troponin was ordered by viewing diagnostic test results in the EMR. Since a primary objective of the study was to assess the utility of troponin testing, patients in whom no troponin was ordered in the ED were excluded. The only other exclusion criteria applied was fever ≥ 38.0°C at triage. Patients were not excluded for reporting fevers prior to ED arrival, as long as fever was not the chief complaint (considered a focal complaint) and they were not documented to be febrile upon arrival. We chose to exclude febrile patients because fever strongly suggests infection and much less likely ACS. Other vital sign abnormalities were permitted.

Since the determination of "nonspecific" is not entirely objective, the first month of patient charts were reviewed by two researchers independently. Agreement about inclusion and exclusion criteria was measured and reported as a kappa value. Discrepancies were resolved through discussion. If agreement was sufficiently high, the remaining charts were to be divided between the two researchers for assessment of inclusion and exclusion criteria.

Once inclusion criteria were verified, additional objective data were abstracted from the EMR using a standardized data collection form, including age, sex, triage chief complaint, admission status, initial troponin positive or negative, results of any subsequent troponin testing during the index visit, and whether or not the patient was documented to have chest pain or shortness of breath on review of systems. Troponin positive was strictly defined according to the institutional cutoffs. The index visit included the ED visit and hospital stay if the patient was admitted. Only the ED visit was counted as index if the patient was discharged from the ED. Abstraction of outcome data and ACS determination were performed by two different physician researchers, as described below.

Outcomes

The outcomes of interest were: 1) the proportion of patients with verified nonspecific complaints who underwent troponin testing; 2) the proportion of such patients who had elevated troponin; 3) the proportion of patients with ACS at the index visit or within 30 days; 4) the utility of troponin testing to diagnose or exclude ACS; and 5) the frequency of other causes of troponin elevation in this population.

During the time period studied, two different troponin assays were utilized. A troponin I point of care whole blood assay (istat, Abbott) with cutoff of 0.08, based on 99th percentile, was primarily used in the ED. Inpatient troponin testing was performed with a troponin I fourth generation (Access, Beckman Coulter). The cutoff is 0.04, also based on 99th percentile. To avoid any subjectivity, we strictly applied the institutional cutoffs in all patients.

Adjudication of ACS

Using the 2014 American Heart Association (AHA) definition for ACS as the standard, we defined ACS as acute myocardial ischemia caused by a partial or complete occlusion of a coronary artery.[20] We further specified ACS as not secondary to noncoronary factors such as demand ischemia or hypoperfusion from sepsis or anemia. To assess for the presence of ACS, each chart was reviewed independently by two trained physician researchers who were blind to the hypothesis and all other aspects of the study. Criteria for the diagnosis of ACS were predefined: 1) any documented ST-elevation myocardial infarction; 2) any coronary revascularization procedure or anatomic test showing acute occlusion or stenosis $\geq$ 70%; 3) stress test or echocardiogram (ECHO) read as consistent with inducible ischemia unless troponins negative and anatomic testing showed no flow restricting lesion; 4) troponin rise and fall in a pattern typical of ACS without an obvious alternative cause (e.g., sepsis, pulmonary embolism). This definition generally conforms to the AHA definition of ACS. Of note, unstable angina (cardiac chest pain without elevation in biomarkers) is considered ACS by the AHA. Our population, by definition, did not have cardiac chest pain, so we limited the diagnosis of ACS to objective findings consistent with cardiac ischemia. Non-ACS causes of troponin elevation were assigned based off the primary team or cardiology team's explanation of troponin elevation.

Agreement was measured and reported. Disagreements were adjudicated through discussion. For any deaths within 30 days, the cause was determined through chart review performed independently by two physicians blinded to the hypothesis of the study. Cause of death, as defined by the clinical team in the discharge or death note, was generally assumed to be accurate. Disagreements were resolved by discussion.

Thirty-day follow-up included a search of the institutional EMR as well as a regional database of medical records compiled from all of the major regional hospital systems (Indiana Healthcare Information Exchange, Careweb). This database includes laboratory results, physicians' notes, discharge summaries, and operative reports from each of the primary hospital systems in the area.

Data Analysis

We assessed the diagnostic utility of troponin as a test for ACS by calculating sensitivity, specificity, negative and positive predictive values (NPV, PPV), and likelihood ratios. We explored how troponin testing performed based on a single blood draw using the first troponin drawn in the ED, as well as the performance accounting for all troponin draws during the index visit, counting any elevated troponin above the institutional cutoff as a "positive" test.

The main objectives were to calculate a rate of ACS in the population, which was assumed to be low, and a false-positive rate (specificity) of troponin testing. As there is no prior literature from which to define the rate of ACS in the population being studied, an assumption based on clinical experience was made that the ACS rate was unlikely to be greater than 2%. To target a 95% confidence interval (CI) with a width of ≤3%, assuming an ACS rate of 2%, approximately 375 patients would be required to provide a 95% CI between 1 and 4%. Assuming a specificity of 90% for troponin, 375 patients would allow for a CI of 86% to 93% for specificity, which was felt unlikely to be viewed as clinically important (i.e., providers would not change their use of the test based on the difference between 86 and 93%).

RESULTS

Patient Identification and Characteristics

The EMR search identified 1,146 unique encounters of ED patients aged 65 and older with nonspecific triage chief complaints between January 1, 2017, and June 30, 2017. After the first 195 encounters (all of January) had been assessed for inclusion by two authors, agreement was 90% ($\kappa = 0.79$, 95% CI = 0.74–0.90). To improve agreement going forward, two additional clarifications were made; dizziness was counted as a nonspecific complaint, regardless of how it was described in the chart (vertiginous versus other) and "troponin ordered in the ED" was defined as drawn within 1 hour of the first blood draw done in the ED. After these clarifications, 100 additional charts were assessed, with 100% agreement. The remaining charts were divided between the two authors for eligibility screening.

Figure 1 outlines the flow of patient identification and exclusions. Of 1,146 encounters, 515 were excluded for having a focal chief complaint upon review of the ED physician note. Thirty-seven patients were then excluded for triage temperature ≥ 38.0°C. Of the remaining 594 patients, 412 (69%) had a troponin drawn in the ED and were included in the study population.

Baseline characteristics for the study cohort are shown in Table 1. The mean (±SD) age was 78.7 (±8.3), and 75% of patients were admitted. Eighty-two patients (20%) had at least one elevated troponin at some point during the index hospitalization. Of these, 52 (63%) had troponin elevation on the first draw in the ED and 30 (37%) had an initially negative troponin. Patients with elevated troponin were more likely to be admitted; more likely to present with a chief complaint of altered mental status; and less likely to present with dizziness, weakness, or fatigue (Table 1).

Main Results

Five patients (1.2%) were determined to have had ACS within 30 days. Details of these patients' courses are provided in Table 2. All cases were identified during the index hospitalization, and no patient developed ACS after being discharged. Agreement for adjudication of ACS was 99.5%. Only one patient was taken for cardiac catheterization and was found to have diffuse coronary disease that was not amenable to intervention. This patient subsequently had a

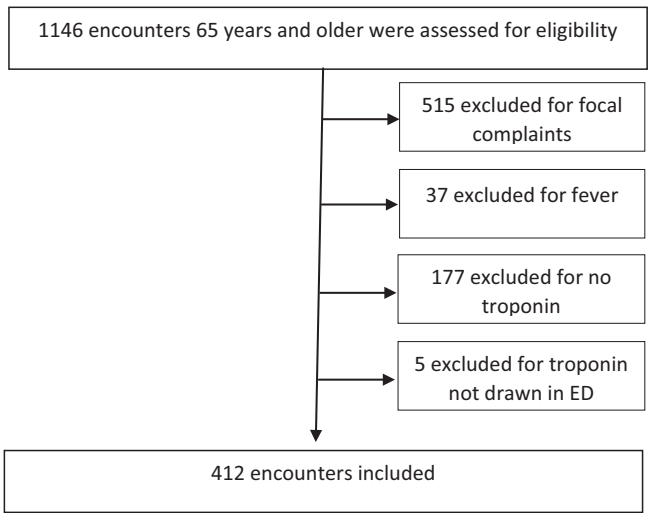

Figure 1. Patient flow chart.

Table 1
Cohort Demographics

	All Patients	Positive Troponin	Negative Troponin
Number of patients	412	82	330
Age (years), mean (±SD)	78.7 (±8.3)	78.8 (±9.4)	78.7 (±8.1)
Admitted, *n* (%)	311 (75)	82 (100)	228 (69)
Female sex, *n* (%)	239 (58)	38 (46)	201 (61)
Chief complaint (%)			
Altered mental status	43	60	39
Weakness/fatigue	33	21	36
Dizziness	21	15	23
Examination requested	2	4	2
Other	<1	<1	<1

cardiac arrest likely secondary to an acute myocardial infarction and was the only patient who died of ACS. No other patients underwent any attempt at reperfusion or had ACS therapy beyond aspirin. The other four ACS diagnoses were all based on troponin elevations without any further formal ACS workup besides nonstress echocardiography.

Counting any elevation as a positive, troponin was 100% sensitive (95% CI = 48%–100%) and 81% specific (95% CI = 77%–85%). The NPV was 100%, and PPV was 6.1%. The positive likelihood ratio (LR+) was 5.26, and the negative likelihood ratio (LR–) was 0. The first troponin drawn in the ED in isolation was 80% sensitive and 88% specific

Table 2
Details of Five Patients With ACS

Patient	Chief Complaint	Peak Troponin (ng/mL)	Imaging for ACS	Discharge Diagnoses	ACS Diagnosis by Clinical Team?	Narrative
82 y/o F	Weakness	>73.0	Angiography showing severe coronary disease and acute thrombus in obtuse marginal vessel	NSTEMI, acute kidney injury, septic shock, pneumonia, hyperkalemia, respiratory failure, ventricular tachycardia	Yes	In ED seen for weakness, diagnosed with hyperkalemia, pneumonia, elevated trop. Inpatient had sepsis requiring vasopressors, troponin peaked at 9, angiography with small acute thrombus. Recovered, off vasopressors, then suddenly developed recurrent arrest and trop > 73, care withdrawn.
92 y/o F	AMS	6.90	ECHO with no WMA, EF 56%	NSTEMI, cerebral contusion, delirium, dementia	Yes	Found confused with evidence of fall, small parietal contusion. No further ACS assessment due to goals of care.
82 y/o F	Dizziness	1.42	ECHO with no WMA	Dizziness, chest pain, troponin elevation medication reaction	No	Seen for dizziness, with subacute CP and SOB on ROS. Troponin 0 in ED. Got fentanyl and desaturated to 45%, recovered with naloxone and sternal rub. Troponin then rose up to 1.4, then down. Treating team felt troponin elevation was due to sternal rub. No cardiology consult.
96 y/o M	Examination requested	3.24	ECHO with no WMA	NSTEMI, unresponsive episode, paroxysmal atrial fibrillation	Yes	Unresponsive episode resolved by ED arrival. Systolic blood pressure 70s in the field. Diagnosed with NSTEMI and transient AMS, no further workup due to goals of care.
78 y/o M	AMS	0.10	ECHO with possible RCA distribution WMA	Unresponsive episode, "CAD with slight trop elevation," multiple myeloma.	No	Brought to ED with brief unresponsive episode. Resolved and workup negative. After ECHO, patient deemed poor candidate for angiography due to multiple myeloma, no further workup pursued.

ACS = acute coronary syndrome; AMS = altered mental status; CAD = coronary artery disease; ECHO = echocardiogram; EF = ejection fraction; NSTEMI = non-ST-elevation myocardial infarction; RCA = right coronary artery; WMA = wall motion abnormalities.

(NPV = 99.7%, PPV = 7.7%). The LR+ was 6.67, and LR– was 0.23.

Neither chest pain nor shortness of breath on review of systems was associated with either troponin elevation or ACS. Thirteen patients were positive for chest pain on review of systems; two had troponin elevation at index and none had ACS. Thirty patients were positive for shortness of breath on review of systems; nine had troponin elevation and none had ACS.

Sepsis was the most common cause of troponin elevation. Table 3 lists all the causes of non-ACS troponin elevation during index visit. Thirty-two patients (7.8%) died within the 30-day follow-up, mostly from sepsis. One patient died of ACS. Table 4 lists all causes of death. Mortality was 19.5% (16/82) in patients with troponin elevation and 4.8% (16/330) in patients with normal troponin. The relative risk for death with elevated troponin was 4.0 (95% CI = 2.0–8.1).

DISCUSSION

In this series of elderly patients presenting to the ED with nonspecific complaints, most patients underwent troponin testing, and although 20% of those tested had an elevated troponin, the diagnostic yield for ACS was low. Only 1.2% of patients (6.0% of those with elevated troponin) were determined to have ACS. Further, only one patient underwent angiography, and no patients received reperfusion therapy.

To our knowledge this is the first study to define the rate of ACS and evaluate the utility of troponin testing in elderly patients with nonspecific complaints. Previous work has found that elderly patients, especially those patients older than 75, with ACS can present with nonspecific complaints.[17,21–23] No previous studies, however, have described the percentage of elderly patients with nonspecific complaints who are diagnosed with ACS. Instead, previous studies, such as the GRACE registry, have examined the percentage of elderly patients with specific complaints, such as vomiting, shortness breath, syncope, and diaphoresis, who had ACS.[23] On the other hand, studies that have examined nonspecific complaints such as weakness have started with a cohort of patients diagnosed with ACS and reported how many ACS patients presented with nonspecific complaints.[23–25] Small series of patients with nonspecific complaints have not reported ACS as an individual diagnosis, instead bundling "circulatory system" problems together.[6,8] This makes comparisons of the rate of ACS with the current study impossible. Our study provides an estimate of the rate of ACS given nonspecific complaints, providing a previously unknown baseline risk estimate or pretest probability in this population. We would note that, for several reasons outlined in the limitations section, the 1.2% risk of ACS in this population may be a conservatively high estimate.

Table 3
Non-ACS Reasons for Troponin Elevation During Initial Visit

Sepsis	22
Dehydration	7
Heart failure	6
Atrial fibrillation	5
Hypertensive emergency	5
Acute respiratory failure	4
Cerebrovascular accident	4
Acute kidney injury	3
Hypotension	3
Anemia	2
Cardiogenic shock	2
Hyperosmolar hyperglycemic state	2
Pulmonary embolism	2
Ventricular tachycardia	2
Adrenal crisis	1
Aortic stenosis	1
Diabetic ketoacidosis	1
Alcohol withdrawal	1
Gastrointestinal bleeding	1
Influenza	1
Seizure	1
Supraventricular tachycardia	1
Sum	77

ACS = acute coronary syndrome.

Table 4
Causes of Death Within 30 Days

Sepsis	11
Cerebrovascular accident	5
Unknown	4
Cancer	2
Cardiogenic shock	2
Failure to thrive	2
Heart failure	2
Colchicine toxicity	1
Gastrointestinal bleed	1
Liver failure	1
Pulmonary embolism	1
Sum	32

There is a wide body of literature demonstrating that troponin elevation predicts worse outcomes in a variety of noncardiac conditions. We found a raw association between troponin elevation and mortality that was very similar to previous works.[26] Since our intention was not to explore troponin as a prognostic marker, we did not collect other prognostic information necessary to perform analyses to calculate whether troponin was an independent predictor of death. We are thus unable to comment on whether troponin added valuable prognostic information on top of clinical judgment and other markers of poor prognosis.

Since there are elderly patients with nonspecific complaints who ultimately have ACS, routine troponin testing might seem like the safest option to avoid missing this important diagnosis. However, in terms of attempting to diagnose ACS, our study did not suggest any patient benefit with this strategy, and there are likely risks that are difficult to quantify. Of the five patients determined to have ACS, four did not undergo any formal testing beyond troponins and nonstress echocardiography, generally due to limited goals of care. The only patient to undergo angiography did not receive reperfusion therapy and ultimately died in the hospital. Although in our study, it seems unlikely that any patient actually benefitted from ACS testing, the elderly population is a very heterogenous one. Our study's patients may have different goals of care than other populations, and it is possible that other elderly patients may request and benefit from more aggressive treatment.

Given the unclear benefit, the potential harm of routine troponin testing in this population should be considered. All 82 patients who had troponin elevation during their index visit were admitted, and almost all received cardiology consultations. Multiple studies have demonstrated a high rate of adverse events in elderly patients who are hospitalized, including delirium and somnolence or nosocomial infections such as pneumonias and urinary tract infections.[27–29] While coronary angiography can benefit elderly patients with diagnosed ACS,[30–33] and should probably be pursued in certain cases, it is a higher risk procedure in this age group.[34,35] Some elderly patients with nonspecific complaints would no doubt benefit from the identification and treatment of ACS, but our findings suggest that such cases are rare. Rather than routine troponin testing, we suggest that the risks, costs, and consequences of downstream testing should be weighed against the potential for benefit and the likelihood of ACS prior to initiating troponin testing in these patients, especially given a false-positive rate of almost 20%.

LIMITATIONS

There are several important limitations to our study. This was a descriptive study with no comparison group, so our findings cannot clearly determine whether liberal troponin testing is associated with any change in admissions, downstream testing, or patient outcomes. Although all patients with troponin elevation were admitted, this should not be interpreted as a causal relationship. The admission rate was high even in patients without troponin elevation, and it is possible that all patients admitted with troponin elevation had other markers.

The determination of what constitutes a "nonspecific" complaint is not entirely objective, and we may have included patients who actually had focal complaints. We attempted to minimize this risk by using triage complaint as a screening tool only and confirming a nonspecific presentation in the providers' notes. Chart reviewers were blinded to whether a troponin had been drawn to limit inclusion bias, and we used two independent reviewers to try to ensure that the determination of a "nonspecific complaint" was reproducible.

Alternatively, our screening criteria could have missed patients with nonspecific complaints who did not present with a triage complaint identified by our EMR search. We consulted with a systemwide IT expert with emergency medicine expertise to ensure that our screening included all triage complaints that would meet the spirit of the study, and we believe that any such misses were rare.

As this was a retrospective chart review, troponin was drawn entirely at the providers' discretion. Since a primary goal of the study was to determine the utility of troponin testing, only patients who underwent troponin testing were included. There were 182 patients with nonspecific complaints excluded for not having a troponin drawn in the ED. Since patients who underwent troponin testing were likely deemed higher risk for ACS by the treating clinician than those who did not, inclusion of these additional patients may have diluted the overall prevalence of ACS, which was only 1.2% among those with troponin testing.

Another limitation of the retrospective nature of the study is that it is impossible to determine why troponin testing was ordered. We have generally assumed that troponin testing is undertaken to assess for ACS, but in some cases, it may have been drawn for prognostic purposes rather than significant concern for ACS.

There are no clear diagnostic criteria for ACS in this population.[36,37] The diagnostic criteria we set forth included troponin elevation as one method to define ACS, even in the absence of other confirmatory testing, as long as no other clear cause for troponin elevation was identified. We tried to minimize the impact of this limitation by blinding the adjudicators of ACS to the hypothesis of the study and defining what constitutes ACS objectively. Nonetheless, the incorporation of the test we were evaluating (troponin) into the criteria for diagnosing ACS may have artificially increased the apparent diagnostic accuracy of the test. This may have played into our results substantially, as four of the five patients diagnosed with ACS had no confirmatory testing beyond abnormal troponins and nonstress echocardiography. Further, two of the five were not felt by their treating clinicians to have ACS. The end result of this limitation is that our finding of a 1.2% ACS rate in this population may have been an overestimation.

Cases of ACS occurring within 30 days but after the initial hospitalization could have been missed. In addition to our institutional EMR, we searched the regional combined EMR database (IHIE Careweb) for cardiology reports and discharge summaries to ensure catchment of any patients diagnosed with ACS at any of the major hospitals within the city and surrounding areas, but diagnoses occurring outside of the regional centers could have been missed.

Finally, this was a single-center study. There could be patient differences in other settings and the threshold for troponin testing could differ in other practice environments.

CONCLUSIONS

Elderly patients with nonspecific complaints in the ED underwent frequent troponin testing, and 20% of those tested had elevated troponin. However, our findings suggest that acute coronary syndrome is rare in this population and the vast majority of patients with elevated troponin do not have acute coronary syndrome. No patients in this study underwent reperfusion therapy. Given the false-positive rate in our study, our results may not support routine troponin testing for acute coronary syndrome in this population.

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