‘Chest Pain Typicality’ in Suspected Acute Coronary Syndromes and the Impact of Clinical Experience

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ABSTRACT

BACKGROUND: Physicians rely upon chest pain history to make management decisions in patients with suspected acute coronary syndromes, particularly where the diagnosis is not immediately apparent through electrocardiography and troponin testing. The objective of this study was to establish the discriminatory value of “typicality of chest pain” and the effect of clinician experience, for the prediction of acute myocardial infarction and presence of significant coronary artery disease.

METHODS: This prospective single-center observational study was undertaken in a UK General Hospital emergency department. We recruited consecutive adults with chest pain and a nondiagnostic electrocardiogram, for whom the treating physician determined that delayed troponin testing was necessary. Using their own clinical judgment, physicians recorded whether the chest pain described was typical or atypical for acute coronary syndrome. Physicians were defined as “experienced” or “novice” according to postgraduate experience. Acute myocardial infarction was adjudicated using a high-sensitivity troponin (hs-cTn) assay, whereas coronary artery disease was adjudicated angiographically.

RESULTS: Overall, 912 patients had typicality of chest pain assessed, of whom 114/912 (12.5%) had an acute myocardial infarction and 157/912 (17.2%) underwent angiography. In patients undergoing angiography, 90/157 (57.3%) had hs-cTn elevation, of whom 60 (66.7%) had significant coronary artery disease. Sixty-seven of 157 (42.7%) patients had angiography without hs-cTn elevation; of these, 31 (46.2%) had significant coronary artery disease. For the diagnosis of acute myocardial infarction, chest pain typicality had an area under the curve (AUC) of 0.54 (95% confidence interval [CI], 0.49-0.60). For the prediction of significant coronary artery disease with hs-cTn elevation AUC: 0.54 (95% CI, 0.40-0.67), and without hs-cTn elevation AUC: 0.45 (95% CI, 0.31-0.59). When assessed by experienced physicians, specificity for the diagnosis of acute myocardial infarction was higher at 65.8% (95% CI, 63.1%-68.7%) vs 55.4% (95% CI, 53.9%-56.8%) for novices.

CONCLUSIONS: Subjective interpretation of “typicality of chest pain” is of limited discriminatory value in the assessment of suspected acute coronary syndromes, in the context of a nondiagnostic electrocardiogram. Greater clinical experience improves accuracy as a rule-in tool but does not improve overall discriminatory ability.

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KEYWORDS: Acute coronary syndrome; Chest pain; Clinical experience; Coronary artery disease; Emergency department; High-sensitivity troponin; Myocardial infarction

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Patients with chest pain symptoms suggestive of acute coronary syndromes account for 10% of all emergency department attendances.\(^1\) Yet, only 15%-20% have a final diagnosis of acute coronary syndrome.\(^2\) In the remainder, distinguishing whether a patient presenting with chest pain has an acute coronary syndrome or a non cardiac problem is difficult.\(^3\) While some alternative diagnoses become apparent using history alone, the ongoing diagnostic uncertainty in the remainder of patients has been shown to lead to emergency department overcrowding, higher levels of resource use, and increased health care costs.\(^4\)\(^-\)\(^6\)

In patients with chest pain and potential acute coronary syndrome, clinical assessment includes electrocardiography and the results of cardiac troponin testing.\(^7\) However, over half of patients will have a nondiagnostic electrocardiogram (ECG),\(^2\) and at least 10% with unstable angina will not have troponin elevations, even in the era of high-sensitivity assays.\(^8\) This sizeable group of patients represents a significant diagnostic and resource challenge. In these situations, physicians often use the perceived discriminatory value of the chest pain history. This will assist in deciding whether a patient is more or less likely to have cardiac chest pain, and the subsequent need for further observation and investigation.\(^9\)

Published data demonstrate that typical symptoms provide useful diagnostic information in patients with stable coronary artery disease.\(^10\)\(^-\)\(^12\) In contrast, evidence examining the value of typical symptoms in patients presenting with chest pain in the acute setting have demonstrated a poor correlation with the final diagnosis of acute coronary syndrome.\(^3\)\(^,\)\(^13\)\(^-\)\(^19\) However, these studies have tended to either include patients with diagnostic ECG changes, focus on specific chest pain characteristics rather than overall typicality, or use research nurses to extract this information. Although it is evident that unstructured clinical judgment, or gestalt, has an important role to play in the overall risk assessment of acute chest pain patients,\(^20\) the subjective interpretation of typicality of chest pain and the impact that clinical experience may have upon its diagnostic accuracy remain poorly understood.

This study therefore aimed to establish, in patients presenting with chest pain and potential acute coronary syndrome with a nondiagnostic ECG, the discriminatory value of “typicality of chest pain” and the effect of clinical experience, for the prediction of acute myocardial infarction and presence of significant coronary artery disease.

**METHODS**

**Study Design and Setting**

This was a planned substudy of a single-center prospective diagnostic cohort study undertaken from July 2012 to August 2013. This study was designed to assess physician risk assessment of emergency department patients with chest pain using an accelerated diagnostic pathway, the results of which have been published previously.\(^21\) It was approved by the UK National Research Ethics Service, registered with the Controlled Trials Database (ISRCTN No. 21109279), and designed using the Standards for Reporting Diagnostic Accuracy.\(^22\) All patient participants provided written informed consent. The study institution’s Emergency Department was situated within a UK District General Hospital and has approximately 62,000 new patient attendances per year.

**Selection of Participants**

We recruited consecutive adults of at least 18 years of age, who had a primary complaint of chest pain, and for whom the treating physician in the Emergency Department determined that delayed (6 hours post attendance) troponin testing was required for the assessment of an acute coronary syndrome. Clinical protocols at the time of the study did not include troponin testing at presentation, unless >12 hours had elapsed since peak symptoms, therefore, assessing physicians were blinded to initial troponin results. In order to focus upon the patient group that provides the greatest diagnostic challenge to physicians, patients were recruited only if they had a nondiagnostic ECG. Patients who were discharged directly from the emergency department at the discretion of the treating physician without delayed troponin testing were not recruited. All patients who required 6-hour troponin testing were admitted to an inpatient assessment unit under the care of an acute general internist; the decision to admit was at the discretion of the assessing physician in the emergency department. Onward cardiology consultation, stress testing, or discharge for outpatient follow-up was at the discretion of the acute internist. Referral for invasive angiography was at the discretion of cardiologists. Recruitment was undertaken 24 hours a day, 7 days a week. Patients were screened by a dedicated researcher and assessed for eligibility and consented in collaboration with the treating physician. Patients were excluded if any of the following were present: ST-segment elevation myocardial infarction or left bundle branch block not known to be old, ECG changes diagnostic of ischemia (ST-segment...
depression ≥1 mm or T-wave inversion consistent with the presence of ischemia), arrhythmias (new-onset atrial fibrillation, atrial flutter, sustained supraventricular tachycardia, second-degree or complete heart block, or sustained or recurrent ventricular arrhythmias), troponin not suitable for analysis (eg, hemolysis), age ≥80 years, a clear nonacute coronary syndrome cause for chest pain was found at presentation (eg, pulmonary embolism, pneumonia, aortic dissection), another medical condition requiring hospital admission, refusal or inability to give informed consent, non-English-speaking, pregnancy, renal failure requiring dialysis, or inability to be contacted after discharge.

Methods and Measurements

On-duty Board Certified Attendings, Senior Emergency Medicine Residents, and Junior Residents all undertook assessments of pain typicality during the study period and completed standardized data collection forms after genuine clinical consultations and before obtaining results of diagnostic tests (other than the initial ECG). To standardize recruitment, ECG evaluation was undertaken by members of the research team. A trained researcher reviewed the hospital record to collect data on the level of clinical experience ascribed to the chest pain described was typical cardiac chest pain in a patient with ischemic symptoms or signs (ECG changes or imaging evidence) would satisfy the diagnosis.25 Based on current consensus guidance for high-sensitivity troponin assays, an increase or decrease of 20% (delta) was considered statistically significant and consistent with a diagnosis of acute myocardial infarction.26 Adjudication of this endpoint was carried out by 2 local cardiologists blinded to the physician interpretation of typical pain, but whom had access to the in-hospital clinical record, General Practitioner records, ECG, troponin, and angiography results.

In order to overcome the diagnostic adjudication challenges associated with high-sensitivity troponin assays, such as small elevations in troponin,27 and evaluate those patients without troponin elevation that have clinically relevant coronary artery disease, we also included a secondary diagnostic outcome measure for those patients assessed angiographically. This was categorized into the presence of significant coronary artery disease with or without high-sensitivity troponin-T elevation (hs-cTnT ≥14 ng/L at either presentation or 6 hours later vs hs-cTnT <14 ng/L at presentation and 6 hours later). Significant coronary artery disease was defined as ≥70% luminal diameter narrowing of at least one major coronary artery as reported on visual assessment by the operator.

Analysis

As this was a planned substudy, no formal power calculation was undertaken, however, previous observational studies reporting the diagnostic utility of chest pain characteristics have typically recruited upwards of 400 participants.13,14,16-19 Chi-squared analyses were used to generate 2 × 2 tables for the calculation of sensitivity and specificity; 95% confidence intervals (CI) are reported. Receiver-operating characteristic curves were obtained by plotting sensitivity against 1-specificity. The area under the receiver-operating characteristic curve (AUC) was chosen as the primary measure of discriminatory value, as it gives a global measure of diagnostic test performance. The AUC was tested against the null hypothesis that typicality of chest pain has no discriminatory ability in determining the presence or absence of acute myocardial infarction or significant coronary artery disease, and therefore the true AUC was 0.50, with a significance of <.05. AUC equals 0.5 when the diagnostic test corresponds to random chance (null hypothesis) and 1.0 indicates perfect diagnostic accuracy. For

knowledge of either the treating physicians’ interpretation of chest pain typicality or troponin results.

Outcomes

The primary endpoint was the diagnosis of fatal or nonfatal acute myocardial infarction occurring during the index visit. The presence of acute myocardial infarction was defined according to the Third Universal Definition, which states that an increase or decrease in troponin with at least one value above the 99th centile value in the context of a patient with ischemic symptoms or signs (ECG changes or imaging evidence) would satisfy the diagnosis.25

...
analysis of the effect of clinical experience, sensitivity (ability of the test to rule out a condition) and specificity (ability of the test to rule in) were compared. All reported P-values are 2-tailed. Statistical analysis was carried out using SPSS version 20 (IBM, Armonk, NY).

RESULTS

Characteristics of Study Subjects

Overall, 912 patients had typicality of chest pain assessed, of whom 394 (43%) were recorded as having had typical chest pain (Figure 1). Acute myocardial infarction occurred in 114/912 (12.5%), and 157/912 (17.2%) underwent angiographic assessment. In those undergoing angiography, 90 (57.3%) had hs-cTnT elevation, of whom 64 (71.1%) had significant coronary artery disease. In the 67 (42%) patients who had angiography without hs-cTnT elevation, 28 (41.7%) had significant coronary artery disease, of whom 21 (67.7%) required percutaneous coronary intervention.

Of the assessments for typicality of chest pain, 227 (24.9%) were made by experienced emergency department physicians and 685 (75.1%) were made by novices. Table 1 summarizes recruited patient demographics according to physician experience; there were no significant differences in clinical characteristics or outcomes (P > .05 for all). When assessed by experienced physicians, a lower proportion of patients were identified as having typical chest pain when compared with novices (35.2% vs 45.8%, P = .005).

Table 2 demonstrates that there was no significant difference in quantitative pretest probability between provider groups when assessed using the TIMI score (in the absence of troponin results).

Contingency tables showing the occurrence of acute myocardial infarction and significant coronary artery disease, with and without hs-cTn elevation according to the presence or absence of typical chest pain, are shown in the Appendix (available online).

Discriminatory Value of Typicality of Chest Pain

The receiver-operating characteristic curves demonstrating the discriminatory ability of typicality of chest pain as assessed by all physicians in the emergency department, either as a diagnostic tool for acute myocardial infarction, or significant coronary artery disease both with and without hs-cTn elevation, are presented in Figure 2. Below this are also listed the values for AUCs according to specific provider groups. When tested against the null hypothesis that the true AUC is 0.50, the P-value for all providers is > .05, suggesting that typicality of chest pain has limited discriminatory ability in the diagnosis or exclusion of acute myocardial infarction and significant coronary artery disease with or without hs-cTn elevation in this cohort across all providers.

Diagnostic Accuracy and Impact of Clinical Experience

Figure 3 presents the sensitivity and specificity of typicality of chest pain for the diagnosis of acute myocardial infarction and significant coronary artery disease both with and without hs-cTn elevation, according to provider groups. As a rule-in tool,
specificity of typical chest pain for all outcomes, when adjudicated by physicians as a whole, ranged from 51.3%-57.9% (95% CI, 40.8%-70.8%). However, when assessing the ability of typicality of chest pain as a rule-in tool according to physician experience, the specificity for the diagnosis of acute myocardial infarction was higher for experienced physicians, at 65.8% (95% CI, 63.1%-68.7%), compared with novices at 55.4% (95% CI, 53.9%-56.8%). Similarly, in patients with significant coronary artery disease without hs-cTnT elevation, there was a trend toward higher specificity when assessed by experienced physicians: 66.7% (95% CI, 48.8%-89.5%) vs 46.7% (95% CI, 34.8%-58.6%). It is important here to note that this finding was not statistically significant due to wide and overlapping 95% confidence intervals. In patients with significant coronary artery disease and hs-cTnT elevation, there was no difference in test specificity between physician groups: 54.5% (95% CI, 28.8-79.4) vs 53.3% (95% CI, 29.6-75.9).

As a rule-out tool for all outcomes, sensitivity of typicality of chest pain ranged from 39.3%-53.1% (95% CI, 24.7%-60.6%). There was no significant difference in the ability of typicality of chest pain to act as a rule-out tool for any outcome measure when comparing experienced and novice physicians.

**DISCUSSION**

Physicians rely upon chest pain history to make management decisions in patients with suspected acute coronary syndromes,
where the diagnosis is not immediately apparent through ECG and troponin testing. Our results show that in emergency patients being assessed for a potential acute coronary syndrome with a nondiagnostic ECG, where troponin results are not yet available, typicality of chest pain is of limited discriminatory value in the prediction of acute myocardial infarction or the presence of significant coronary artery disease. With regard to clinician experience, the diagnostic accuracy for the rule-in of acute myocardial infarction appears to be greater with more experience, but this difference is small and therefore likely to have limited clinical applicability.

Our report has several important implications. Physicians intuitively adopt a Bayesian approach to diagnosis, making an initial diagnosis based on probabilities, then adjusting these probabilities as more information becomes available. This diagnostic approach is applied readily in the risk assessment of patients with acute chest pain. In the absence of diagnostic ECG changes, physicians weigh up all the information gathered from the history and physical assessment to establish pretest probability before the results of troponin testing. Chest pain typicality has been traditionally central to this assessment, yet our results bring its value into question.

Until recently, contemporary cardiac troponin assays could be used reliably to identify those patients with a non-ischemic ECG who were at high risk for acute myocardial infarction and adverse events with low false-positive rates. However, the development of high-sensitivity cardiac troponin assays, which can detect troponin in over 50% of apparently illness-free individuals, has raised concerns around binary positive and negative interpretation of results. The potential for multiple acute conditions to cause elevations in hs-cTn has necessitated better estimates of pretest probability to allow improved management decisions based on elevated hs-cTn results. Our results suggest that chest pain typicality may also be of limited use in this regard.

With the advent of high-sensitivity assays, there was the potential that in clinical practice, their use may make the diagnosis of unstable angina obsolete. By using hs-cTn to adjudicate our primary endpoint and confirm the findings in a subset of patients who have undergone angiographic assessment, we have demonstrated an important finding. In our cohort, over 40% of
patients assessed angiographically had significant coronary artery disease, in the absence of high-sensitivity troponin elevation, and two-thirds of these required intervention. It is these patients in whom treating physicians most rely on the discriminatory value of the chest pain history, yet we demonstrate that typicality may again be of limited use.

We therefore suggest that there should be a focus on the clinical application of accelerated diagnostic protocols, which combine risk scores with high-sensitivity troponin testing rather than unstructured subjective clinical assessment or gestalt.

Importantly, this study included only patients with chest pain and a potential acute coronary syndrome with a non-diagnostic ECG who were admitted to a ward for delayed biomarker testing. As a result, the treating clinician had already used clinical judgment to identify patients with chest pain in whom there was a high level of suspicion for acute coronary syndrome and therefore required further inpatient evaluation. Those patients with diagnostic ECGs and those discharged directly from the emergency department with “non-concerning” histories were, as a result, intentionally excluded from analysis. Although our population therefore may be subject to significant selection bias, we have focused intentionally on a cohort of patients that provide the greatest diagnostic challenge for acute physicians on a day-to-day basis. It is possible that the discriminatory value of typicality of chest pain would have improved if patients with clinically evident acute coronary syndromes also had been recruited for analysis. However, the exclusion of those patients in whom there was no diagnostic uncertainty has allowed us to provide novel insight into an everyday and highly relevant clinical problem.

There are some limitations to this study. The applicability of the results may be limited by the characteristics of the population selected. The inclusion of predominantly white patients may limit the applicability to international settings, especially as cross-cultural differences in symptom reporting exist. The upper age cut-off of ≥80 years was chosen for pragmatic institutional reasons, as patients were cared for by different inpatient teams. This may limit further the general applicability of the findings, as firstly, older individuals are more likely to report atypical symptoms and secondly, older cohorts are known to have high proportions of patients with elevated hscTn assay results, often due to subclinical disease.

CONCLUSION

Physician interpretation of “typicality of chest pain” is of limited discriminatory value in patients being assessed for potential acute coronary syndromes, in the context of a nondiagnostic ECG. Greater clinical experience improves accuracy as a rule-in tool for acute myocardial infarction, but this does not improve overall discriminatory ability.

References


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**Conflict of Interest:** EC has received funding from Abbott in support for related research. MT has received funding from Alere, Abbott, Beckman, and Roche for speaking and support for other research. LC has received funding from Abbott, Roche, Alere, Siemens, and Radiometer Pacific for clinical trials, and from Alere, Boehringer Ingelheim, Pfizer, Astra Zeneca, Abbott, Novartis, and Radiometer Pacific for speaking and education. AK has no conflicts of interest. KG has received funding from AstraZeneca and Takeda UK for related research.

**Authorship:** Each author has contributed to the analysis and interpretation of the data, drafting and approval of the final manuscript. All authors have also contributed to the conception/design of the study reported in this manuscript.

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**SUPPLEMENTARY DATA**

Supplementary tables accompanying this article can be found in the online version at [http://dx.doi.org/10.1016/j. amjmed.2015.04.012](http://dx.doi.org/10.1016/j. amjmed.2015.04.012).
Contingency tables showing the occurrence of acute myocardial infarction (Supplementary Table 1) and significant coronary artery disease with and without troponin elevation (Supplementary Table 2) according to the presence or absence of typical chest pain.

**Supplementary Table 1**  The occurrence of acute myocardial infarction according to the presence or absence of typical chest pain

<table>
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<th>Acute Myocardial Infarction</th>
<th>No Acute Myocardial Infarction</th>
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<tr>
<td>All physicians</td>
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<tr>
<td>Typical chest pain present</td>
<td>58</td>
<td>336</td>
<td>394</td>
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<tr>
<td>Typical chest pain absent</td>
<td>56</td>
<td>462</td>
<td>518</td>
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<tr>
<td>Total</td>
<td>114</td>
<td>798</td>
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<tr>
<td>Experienced physicians</td>
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<tr>
<td>Typical chest pain present</td>
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<tr>
<td>Total</td>
<td>34</td>
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<td>227</td>
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<tr>
<td>Novice physicians</td>
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<tr>
<td>Typical chest pain present</td>
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<td>270</td>
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<tr>
<td>Total</td>
<td>80</td>
<td>605</td>
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### Supplementary Table 2  
The occurrence of significant coronary artery disease with and without troponin elevation according to the presence or absence of typical chest pain

<table>
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<tr>
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<th>Significant Coronary Artery Disease and hs-cTnT ≥14 ng/L</th>
<th>No Significant Coronary Artery Disease and hs-cTnT ≥14 ng/L</th>
<th>Total</th>
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<tr>
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<td>44</td>
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<td><strong>Experienced physicians</strong></td>
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<tr>
<td><strong>Total</strong></td>
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<tr>
<td><strong>Novice physicians</strong></td>
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<td>19</td>
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<th>Significant Coronary Artery Disease and hs-cTnT &lt;14 ng/L</th>
<th>No Significant Coronary Artery Disease and hs-cTnT &lt;14 ng/L</th>
<th>Total</th>
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<td><strong>All physicians</strong></td>
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<tr>
<td>Typical chest pain present</td>
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<td>19</td>
<td>30</td>
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<tr>
<td>Typical chest pain absent</td>
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<tr>
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<tr>
<td>Typical chest pain absent</td>
<td>8</td>
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<tr>
<td><strong>Total</strong></td>
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<tr>
<td><strong>Novice physicians</strong></td>
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<tr>
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<tr>
<td><strong>Total</strong></td>
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Hs-cTnT = high-sensitivity troponin-T elevation.