

Beyond Chest Pain: Diagnostic Pathway Failures and Time-to-Treatment Disparities in Acute Coronary Syndrome

—A Focused Narrative Review

Zaidan Khawaled^{1*}, Amira Amara²

¹Bnai Zion Medical Center, Haifa, Israel

²Wolfson Medical Center, Holon, Israel

Email: *khawaled.zai@gmail.com

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Abstract

Background: Acute coronary syndrome (ACS) pathways are commonly activated by chest pain, creating a systems vulnerability when myocardial ischaemia presents with dyspnoea, gastrointestinal symptoms, syncope, weakness or fatigue. **Objective:** To examine recent evidence on where recognition fails and how diagnostic and treatment delays can be measured. **Methods:** PubMed/MEDLINE and Google Scholar were searched to July 2026 for adult ACS studies reporting symptom phenotype and triage, diagnostic or treatment intervals. **Results:** Contemporary studies consistently located the largest disparities early in care. In a 2021 PCI cohort, door-to-balloon time within 90 minutes was achieved in 40.0% of atypical presentations compared with 66.3% of typical presentations. A 2024 study found threefold higher odds of delayed reperfusion, while a 2025 STEMI cohort reported markedly higher odds of delayed door-to-ECG time. **Conclusion:** Symptom-inclusive triage, parallel cardiac evaluation and routine auditing of door-to-ECG, door-to-diagnosis and diagnosis-to-treatment intervals according to presenting symptom should be prioritised.

Keywords

Acute Coronary Syndrome, Anginal Equivalent, Diagnostic Delay, Door-to-ECG, Reperfusion

1. Introduction

Time-sensitive evaluation is fundamental to ACS care. Current European and

North American guidance supports rapid electrocardiography (ECG), serial high-sensitivity cardiac troponin testing, risk stratification and timely reperfusion when ACS is suspected [1] [2]. The operational weakness is that suspicion is often generated by the presence of chest pain. Patients whose dominant complaint is dyspnoea, nausea or vomiting, epigastric discomfort, syncope, palpitations, weakness, fatigue or confusion may instead enter respiratory, gastrointestinal, neurological or general medical pathways. Those pathways are clinically reasonable, but sequential organ-specific assessment can defer cardiac testing when myocardial ischaemia is not considered in parallel.

Terminology contributes to this problem. Across 43 studies in a 2025 systematic review, the proportion labelled as having an “atypical” presentation ranged from 4.6% to 74.2%, while presentations explicitly without chest pain ranged from 1.4% to 35.5% [3]. A 2024 scoping review similarly found inconsistent definitions across 41 studies, with dyspnoea the most frequently reported non-chest-pain symptom [4]. The term “atypical” can therefore combine several exposures: absence of chest discomfort, unusual pain descriptors, non-thoracic pain or mixed symptoms. It should not be treated as a diagnosis or a marker of low risk. This review uses “without chest pain” when that exposure was explicitly reported and retains “atypical” only when it was the source study’s definition.

Recent reviews have established symptom diversity, risk factors and adverse outcomes [3] [4]. The novel features in the present review is an operational pathway lens. It acknowledges that non-chest pain presentations are not an event that should trigger ACS care; it prioritises studies that measure objective diagnostic and treatment time intervals and it translates time intervals into auditable quality measures. This is further supported by a 2025 study during PCI era that looked at door to balloon time, which was delayed and a 2026 consensus study that looked at delayed and missed coronary diagnosis. The central question is: Where does preventable delay occur before treatment, beyond the issue of whether mortality is higher?

2. Methods

This focused narrative review was designed to synthesise contemporary evidence on diagnostic pathway failure and measurable treatment delay in adults with ACS or Acute Myocardial Infarction (AMI) presenting without chest pain or with symptoms classified by investigators as atypical. This research involved searching PubMed/MEDLINE and Google Scholar on July 2026. Available lists of eligible reviews and primary studies were reviewed and the websites of the European Society of Cardiology and the American College of Cardiology and the American Heart Association were searched for up-to-date guidelines and consensus pathways.

The PubMed search was: acute coronary syndrome, myocardial infarction, STEMI, NSTEMI AND without chest pain, no chest pain, dyspnoea, anginal equivalent, AND delay, triage, door-to-ECG, door-to-balloon, reperfusion, misdiagno-

sis, treatment. The date filter was 2020 to 2026. Google Scholar used the simplified search phrase: (acute coronary syndrome OR myocardial infarction) (without chest pain OR atypical symptoms) (delay OR triage OR ECG OR reperfusion OR treatment).

Eligible publications were English-language adult cohort studies, registries, systematic or scoping reviews, and major guidelines or consensus documents published from 2020 through 2026. Evidence had to report at least one of the following: a reproducible symptom definition; prehospital response or delay; time to ECG, troponin, diagnosis, catheter-laboratory activation or reperfusion; treatment delivery; or an adjusted outcome linked to symptom presentation. No paediatric studies, case reports, non-ACS populations or reports with no separable data for symptoms or delay were included. One reviewer screened titles and abstracts, a subset of possible full texts was reviewed, and studies were purposefully selected when they focused on a pathway stage or provided an interval or effect estimate that could be directly extracted.

For each included report, the symptom definition, setting, population, care interval, treatment measure and adjusted effect estimate were extracted into a structured evidence table. Findings were synthesised by pathway stage: patient recognition, emergency response, triage, diagnostic testing, escalation and treatment. This was not a systematic review: no protocol was registered, screening and extraction were not duplicated, and formal risk-of-bias or certainty assessment was not performed. No new meta-analysis was undertaken. Numerical values in tables and figures were restricted to directly reported results. **Table 1** summarises the included evidence by population, symptom exposure, direct delay finding and primary pathway message.

Table 1. Contemporary evidence locating delay along the ACS pathway.

Study	Population	Symptom exposure	Direct delay finding	Primary pathway message
DeVon <i>et al.</i> 2020 [7]	Suspected ACS	Unusual fatigue; EMS non-use	Associated with prolonged prehospital delay	Patient recognition and transport
Møller <i>et al.</i> 2021 [8]	8336 MI cases	Chest pain absent versus present	Less emergency dispatch without chest pain	Emergency-response classification
Nakahashi <i>et al.</i> 2021 [12]	1337 AMI patients receiving PCI	11.2% atypical	D2B ≤ 90 min: 40.0% versus 66.3%	Slower reperfusion
Al-Rumhi <i>et al.</i> 2024 [11]	426 STEMI patients	Atypical versus typical	Delayed D2B OR 3.003 (95% CI 1.409 - 6.400)	Triage and organisational delay
Huang <i>et al.</i> 2025 [10]	807 STEMI patients receiving PCI	13.5% atypical	Door-to-ECG aOR 11.52; later DTB components also delayed	Largest gap occurs before ECG

Continued

Van Schalkwijk <i>et al.</i> 2026 [15]	Modified Delphi consensus	ACS diagnostic process	Time-bounded definitions for delayed and missed diagnosis	Standardised audit thresholds
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ACS, Acute Coronary Syndrome; AMI, Acute Myocardial Infarction; aOR, adjusted Odds Ratio; CI, Confidence Interval; D2B, Door-To-Balloon; ECG, Electrocardiography; EMS, Emergency Medical Services; MI, Myocardial Infarction; OR, Odds Ratio; PCI, Percutaneous Coronary Intervention; STEMI, ST-Segment Elevation Myocardial Infarction.

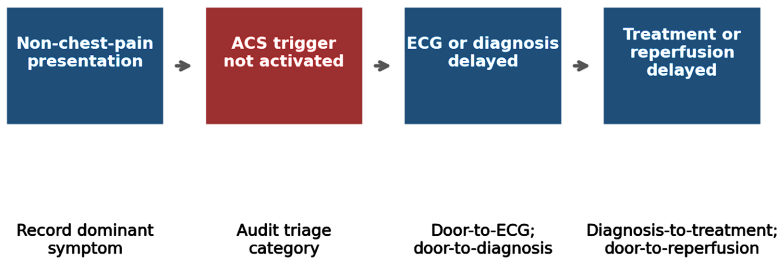
2.1. Phenotype and Pathway Vulnerability

Absence of chest pain is more reproducible than the broad label “atypical”, although presentations remain heterogeneous. Symptoms include dyspnoea, nausea, epigastric or back discomfort, diaphoresis, syncope, palpitations and fatigue. These are plausible manifestations of ventricular dysfunction, autonomic activation, reduced cardiac output or ischaemia-related arrhythmia.

Older age, diabetes, heart failure, cerebrovascular disease, haemodynamic instability and NSTEMI are associated with non-chest-pain presentation [3] [4]. Women may report a broader symptom mix but commonly experience chest discomfort; labelling women inherently “atypical” risks stereotyping. Current guidance recommends specific symptom descriptors [5] and recognises chest, shoulder, arm, neck, back, upper-abdominal or jaw discomfort, dyspnoea and fatigue as possible anginal equivalents [6]. Subgroup information should heighten vigilance, not replace ECG and troponin assessment.

Dominant symptoms can redirect attention to a complication rather than its coronary cause. Dyspnoea may reflect ventricular dysfunction and syncope impaired output or arrhythmia. Respiratory treatment may be necessary, but immediate ECG assessment should proceed in parallel when ACS is plausible. **Figure 1** illustrates how failure to recognise ACS at triage can delay ECG acquisition, diagnosis, escalation and reperfusion.

A recognition failure at triage propagates through the entire ACS pathway



Recommended response: symptom-inclusive triggers and parallel cardiac evaluation

Figure 1. Diagnostic pathway model. A failure to recognise ACS at triage delays ECG and diagnosis, with downstream effects on escalation and reperfusion. ACS, acute coronary syndrome; ECG, electrocardiography.

2.2. Where Time Is Lost

Patient-level recognition is the first potential failure. Symptoms such as fatigue, indigestion or breathlessness may be attributed to infection, anxiety, gastrointestinal illness, chronic lung disease or ageing. In a prospective study of suspected ACS, unusual fatigue and failure to use emergency medical services were associated with prolonged prehospital delay [7]. A Danish study of 8336 patients with MI linked symptom presentation to emergency response: patients without chest pain were less likely to receive an emergency dispatch and had worse short-term survival [8]. A systematic review from low- and middle-income countries found that delay reflects interacting patient, transport and health-system barriers, underscoring the importance of measuring symptom-onset-to-first-medical-contact and first-medical-contact-to-hospital intervals separately [9].

The next failure occurs at triage. A chest-pain-dependent protocol can place a patient with dyspnoea in a respiratory stream or a patient with epigastric discomfort in a gastrointestinal stream without a concurrent 12-lead ECG. The 2025 cohort by Huang *et al.* provides the clearest contemporary decomposition of this delay. Among 807 patients with STEMI undergoing primary PCI, 13.5% had atypical symptoms. Atypical presentation was associated with delayed door-to-ECG time (adjusted odds ratio [aOR] 11.52, 95% CI 6.04 - 22.06), activation-to-catheter-laboratory arrival (aOR 1.71, 95% CI 1.04 - 2.80) and catheter-laboratory-arrival-to-balloon time (aOR 1.95, 95% CI 1.24 - 3.06) [10]. The magnitude and sequence of these estimates suggest that the largest inequity emerges before the diagnosis is secure, then propagates downstream. **Figure 2** displays these adjusted delay estimates.

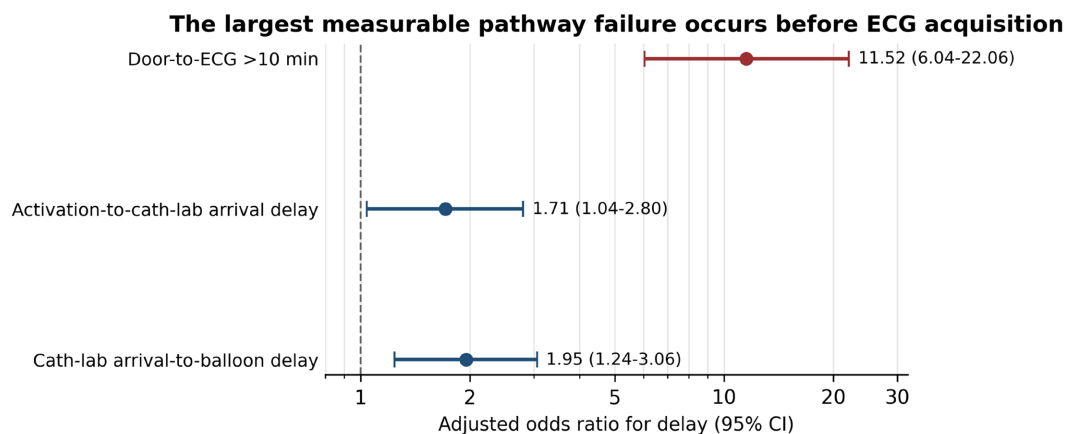


Figure 2. Adjusted odds of delay with atypical versus typical STEMI presentation in the 2025 PCI-era cohort by Huang *et al.* [10]. The largest association was for door-to-ECG time exceeding 10 minutes. CI, confidence interval; ECG, electrocardiography; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

Early delay has three modifiable mechanisms. Pattern recognition fails when symptoms do not match the chest-pain script; triage templates may use chest discomfort as the ECG gateway; and a plausible alternative diagnosis can cause an-

choring. Education can broaden clinical scripts, electronic rules can widen ECG eligibility, and team prompts can mandate reassessment when vital signs, troponin or the clinical course conflict with the initial diagnosis.

Other PCI-era studies support this mechanism. In a 2024 Omani cohort, atypical symptoms were associated with approximately threefold higher odds of delayed door-to-balloon time (OR 3.003, 95% CI 1.409 - 6.400) [11]. A 2021 AMI registry likewise found less timely reperfusion with atypical presentation (**Figure 3**) and higher 30-day mortality (hazard ratio 2.33, 95% CI 1.20 - 4.38) [12]. Despite different definitions and settings, studies show that patients who do not match the expected presentation move less rapidly through ECG acquisition, escalation and reperfusion.

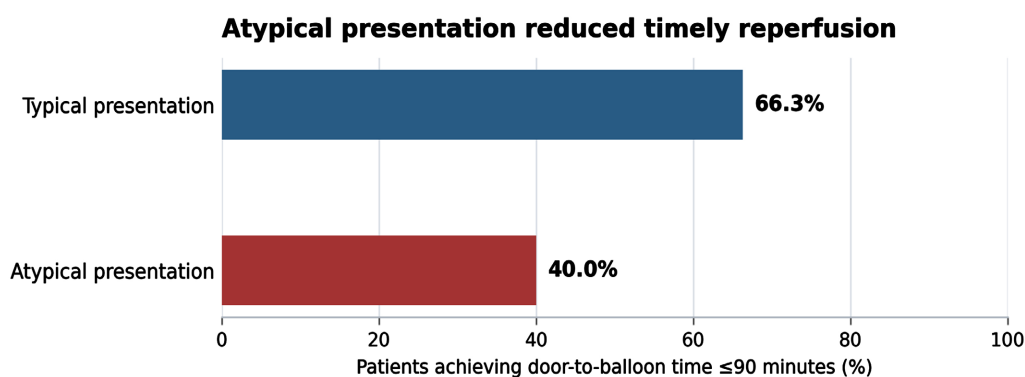


Figure 3. Timely reperfusion by presentation phenotype. Door-to-balloon time within 90 minutes was achieved in 40.0% of atypical versus 66.3% of typical AMI presentations [12]. AMI, acute myocardial infarction; PCI, percutaneous coronary intervention.

A total door-to-balloon interval can conceal delay in ECG acquisition, repeat testing, cardiology review, catheter-laboratory activation, transfer or the procedure. Separating components directs intervention: triage redesign for late ECG, decision support for late escalation, and network coordination for transfer delay. **Figure 1** shows how early recognition failure propagates downstream.

Treatment disparity follows recognition failure. Late ECG or diagnosis can delay or prevent antithrombotic therapy, specialist review, angiography and reperfusion. Recent syntheses report less consistent evidence-based care in non-chest-pain presentations, although many estimates derive from older registries [3] [4]. Modern audits should test whether phenotype predicts treatment omission after adjustment for contraindications, frailty, haemodynamic severity and ACS subtype.

2.3. From Recognition to Treatment

Once ACS is recognised, treatment should follow risk and ECG findings, not pain presence. Guidance recommends immediate ECG, serial high-sensitivity troponin, repeat ECG when suspicion persists, appropriate antithrombotic therapy and urgent STEMI reperfusion [1] [2] [13] [14]. These measures require reliable

pathway entry and parallel evaluation of cardiac and competing diagnoses. A symptom-inclusive trigger can include new unexplained dyspnoea; diaphoresis with nausea or vomiting; exertional epigastric, back, arm or jaw discomfort; acute pulmonary oedema; unexplained hypotension; high-risk syncope; or new arrhythmia with weakness. These findings justify early ECG and possible troponin testing.

To avoid excessive testing, triggers should incorporate age, cardiovascular risk, abnormal vital signs, clinician concern and local prevalence. Electronic prompts should recommend ECG, not diagnose ACS, and be evaluated for sensitivity, workload, crowding and unintended inequity. In haemodynamic instability, acute heart failure or diagnostic uncertainty, bedside echo can be used to aid assessment, but not delay STEMI reperfusion. ECG is not useful as a rule out test for ACS; serial troponin and repeat ECG are still valuable when there is persistent ACS symptoms or a change in clinical status.

The short bedside note should record onset of symptoms and autonomic features, vital signs and perfusion, immediate 12-lead ECG if ACS is considered, and should include troponin and tests for competing emergencies. Set a re-evaluate time point for repeat ECG, troponin review and escalation, if symptoms or instability continues. Implementation involves symptom-inclusive dispatch scripts, nurse-authorised ECG requests, rapid repeat testing, rules for access to cardiology and escalation to the catheter-lab that are not based on pain severity. Teams should review delayed cases and feedback should be provided to them on a chart indicating where delays started in the intervals.

2.4. Measurement and Research Priorities

Quality improvement requires an explicit denominator: chest discomfort present or absent, dominant symptom and mixed presentations. Report medians and interquartile ranges for onset-to-first-medical-contact, door-to-ECG, door-to-troponin, door-to-diagnosis, diagnosis-to-specialist activation and door-to-reperfusion. Also report target attainment as similar medians can mask a high-risk tail of delay. The 2026 Delphi study recommended time-limited definitions of delayed and missed coronary diagnosis such as a missed STEMI if the diagnosis had not been made 90 minutes after arrival [15]. These thresholds enable comparable audit. Validation should test associations with treatment omission, infarct size, heart failure and mortality across emergency systems.

Research should separate STEMI from NSTEMI, absence of chest pain from broad atypical classifications, prehospital from in-hospital delay, and diagnostic from procedural delay. Prospective multicentre cohorts should use time-stamped electronic data and prespecify age, sex, diabetes, renal disease, heart failure, haemodynamic severity, ambulance use and transfer status. Implementation trials should compare usual chest-pain triggers with symptom-inclusive triage rules. The primary process outcome should be door-to-ECG by phenotype, with unnecessary ECGs, false activations, length of stay and clinical outcomes as balancing measures. Report results by age, sex and diabetes.

Analyses should preserve event order by modelling door-to-ECG, diagnosis, activation and reperfusion separately. Mediation analysis can estimate how delayed recognition contributes to treatment delay. Time-to-event and quantile methods can address variable follow-up and delayed tails. Report missing timestamps and uncertain symptom onset, with sensitivity analyses for imputation. Equity analysis should cross-tabulate symptom phenotype with age, sex, diabetes, ambulance use and transfer status while suppressing unstable small-cell estimates. Interviews with patients and triage staff can explain measured gaps and guide acceptable changes to public messaging, triage questions and escalation procedures. **Table 2** presents the proposed minimum audit set.

Table 2. Minimum audit set for ACS presentations without chest pain.

Pathway stage	Required timestamp or variable	Preferred report	Balancing or equity measure
Phenotype	Chest discomfort present/absent; dominant and mixed symptoms	Proportion by explicit phenotype	Missing symptom documentation
Prehospital	Symptom onset; call; first medical contact; hospital arrival	Median, IQR and delayed tail	Ambulance use and transfer status
Diagnosis	Arrival/FMC to first ECG, troponin and confirmed diagnosis	Median, IQR; percentage meeting target	ECG volume and false-positive activation
Escalation	Diagnosis to cardiology review or cath-lab activation	Median, IQR and threshold attainment	After-hours and interhospital variation
Treatment	First antithrombotic; angiography; wire crossing or balloon	Interval by STEMI/NSTEMI and symptom	Age, sex, diabetes and comorbidity

FMC, First Medical Contact; ECG, Electrocardiography; IQR, Interquartile Range; NSTEMI, Non-ST-Segment Elevation Myocardial Infarction; STEMI, ST-Segment Elevation Myocardial Infarction.

3. Strengths and Limitations

This review prioritises care-stage failures, contemporary PCI-era estimates and measurable audit variables, while distinguishing absence of chest pain from source-defined atypical presentation. The limitations are a limited and focused search, screening and extraction of data by a single reviewer, limited database coverage, and not using a formal risk-of-bias assessment. The majority of the delay studies were observational, single country or single centre, employed varied definitions of symptoms and suffered from confounding due to age, comorbidity and symptom severity. No mortality associations imply excess risk due to delay. For NSTEMI, there is less evidence and for STEMI, there is more evidence. Restriction to 2020-2026 was beneficial for currency but had to depend on the recent reviews to get

context for earlier registry.

4. Conclusion

ACS without chest pain is a pathway-recognition problem. Measurable disparities begin before or at ECG acquisition and persist through escalation and reperfusion. The response is symptom-inclusive triage, parallel cardiac evaluation and reporting of door-to-ECG, door-to-diagnosis and diagnosis-to-treatment intervals by phenotype. Implementation trials should test whether redesigned pathway entry reduces delay without excessive testing. Evaluation should also examine diagnostic safety, unnecessary testing, equity and whether benefits persist across STEMI, NSTEMI and different emergency-care settings and diverse international patient groups.

Ethics Approval and Consent to Participate

Not applicable; this article reviews published evidence and reports no new participant data.

Author Contributions

Research and writing the manuscript.

Data Availability

No new dataset was generated. All numerical data are available in the cited publications.

Generative AI Disclosure

OpenAI ChatGPT/Codex was used to support English-language editing and reference-format checking.

Conflicts of Interest

The authors declare no conflicts of interest.

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List of Abbreviations

ACS	Acute Coronary Syndrome
AMI	Acute Myocardial Infarction
aOR	Adjusted Odds Ratio
CI	Confidence Interval
D2B	Door-to-Balloon
ECG	Electrocardiography
EMS	Emergency Medical Services
FMC	First Medical Contact
MI	Myocardial Infarction
NSTEMI	Non-ST-Segment Elevation Myocardial Infarction
OR	Odds Ratio
PCI	Percutaneous Coronary Intervention
STEMI	ST-Segment Elevation Myocardial Infarction