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Standardized emergency department protocol for non-traumatic chest pain: experience from the USL Umbria 1 Health Authority

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Abstract

This document describes the standardized corporate procedure developed by the USL Umbria 1 Health Authority to standardize the multidisciplinary approach to patients presenting with non-traumatic chest pain across its network of emergency departments, a common presentation requiring rapid differentiation between life-threatening conditions [*e.g.*, acute coronary syndrome (ACS), aortic dissection, pulmonary embolism] and benign etiologies. The protocol mandates a structured pathway focusing on early risk stratification, including a 12-lead electrocardiogram (ECG) within 10 minutes of arrival and a comprehensive assessment to determine initial triage priority. For suspected ACS, the procedure involves serial cardiac biomarker (troponin) measurement (T0, T3h, and T6h) and HEART Score calculation to estimate the 30-day risk of major adverse cardiovascular events. High-risk patients (HEART Score 7 or clinically unstable) receive immediate cardiology consultation. Specifically for diagnosed ST-elevation myocardial infarction (STEMI) cases, the protocol integrates with the regional Umbria STEMI Network, ensuring immediate activation of the 118 emergency service for rapid secondary transport from spoke/antenna hospitals to a primary percutaneous coronary intervention (pPCI) hub center, with pPCI being the preferred reperfusion strategy and fibrinolysis reserved only if the time to pPCI exceeds 110-120 minutes from the first medical contact. Bedside echocardiography is an encouraged tool for rapid differential diagnosis and left ventricular function assessment. The implementation of this standardized, evidence-based protocol, integrated with the regional STEMI Network and leveraging rapid ECG teletransmission and risk scoring tools (Chest Pain Score, HEART), aims to ensure timely, guideline-driven, and appropriate care for all patients with non-traumatic chest pain across the USL Umbria 1 Emergency Departments.

Introduction

Cardiovascular disease remains the leading cause of mortality in industrialized nations, with coronary artery disease (CAD) being the most common condition associated with high morbidity and mortality.¹ Non-traumatic chest pain (DT) is a frequent and challenging presentation in the emergency department (ED).¹ Diagnostic difficulty arises from the lack of correlation between symptom intensity and disease severity, and the frequent overlap of symptoms between benign and life-threatening conditions.^{2,3} The clinical presentations of CAD encompass silent ischemia, stable/unstable angina pectoris, acute coronary syndrome (ACS), heart failure, and sudden death.⁴ Notably, non-ST elevation myocardial infarction (NSTEMI) is documented to be significantly more frequent than ST-elevation myocardial infarction (STEMI).

This procedural document was established by the USL Umbria 1 Health Authority to serve as a guideline for managing ED patients with non-traumatic chest pain.⁵ Its primary objective is to guarantee the timely management and correct initiation of treatment for patients presenting with potentially lethal conditions, including ACS, aortic dissection, and pulmonary embolism (PE).⁶

Materials and Methods

The procedure applies to all patients presenting with chest pain to the USL Umbria 1 EDs.⁵ Non-traumatic chest pain (DT) is defined as any pain located anteriorly between the nose and the umbilicus, and posteriorly between the nape and the 12th vertebra, or a symptom equivalent, onset within the 24 hours preceding observation, which has regressed or is still present, and is not attributable to prior trauma or another immediately identifiable cause.⁷

Triage process and initial actions

The triage process is crucial for rapid and accurate evaluation to differentiate life-threatening issues.⁸ **Electrocardiogram (ECG) mandate:** A 12-lead ECG must be promptly performed on all chest pain patients within 10 minutes of arrival. The USL Umbria 1 system utilizes telemedicine for ECG tele-reporting by the on-call cardiologist.⁸

Vital parameters: essential vital signs [blood pressure (BP), heart rate (HR) and oxygen saturation (SpO₂)] and a rapid patient evaluation are mandatory.⁸

Triage coding:⁵ i) red code (immediate access): assigned for typical/atypical chest pain with signs of instability [e.g., SpO₂ <85% in room air, cold sweat, cyanosis, marked dyspnea respiration rate (RR)<10 or >30], or severe brady/tachycardia (HR<40 or >150 bpm)]; ii) orange code (immediate evaluation/advanced triage): assigned for typical chest pain [Chest Pain Score (CPS) ≥ 4] in progress, recent onset in patients with known ischemic heart disease, syncope, age >40 years with cardiovascular risk factors, or severe hypertension [systolic blood pressure (SBP) 180 and diastolic blood pressure (DBP) >110]; iii) blue code (deferred urgency): assigned for atypical, mild (Visual Analogue Scale 1-3), localized, or pleuritic pain, often associated with fever or cough, with normal vital signs and low CPS (<4).

Patient history and Chest Pain Score

A complete history, investigating the nature, onset, duration, location, radiation, precipitating, and relieving factors, and associated symptoms, is essential.⁹ CPS is used to quantify the likelihood of ischemic origin.¹⁰ A score ≥ 4 indicates high risk, and a score >8 is classified as very high risk.⁵ Patients with CPS ≥ 4 must have blood drawn for cardiac markers (troponin) at triage.¹¹

Emergency physician and nurse actions

The ED physician must confirm or exclude an acute coronary cause and consider non-cardiac diagnoses.¹² The evaluation focuses on: i) symptom quality – assessment of CPS; ii) troponin assay – plasma troponin must be measured at T0 (upon presentation). A second measurement is required if the chest pain onset (DT) was hours from T0, or in cases where the initial T0 result shows a borderline elevation. For laboratories or EDs utilising conventional point-of-care testing methods, serial

determinations (T0, T3h, T6h) are required to rule out ACS with high sensitivity; iii) echocardiography – bedside echo is recommended to rapidly assess left ventricular systolic function (a key prognostic variable) and aid in the differential diagnosis of aortic dissection, PE, aortic stenosis, hypertrophic cardiomyopathy, and pericardial effusion.¹³ Nursing actions in the ED include multi-parametric monitoring, obtaining appropriate intravenous access, and administering prescribed therapy.¹⁴

Management of acute coronary syndrome

ST-elevation myocardial infarction management via the Umbria Regional Network

Optimal STEMI management in the Umbria Region is achieved through a consolidated STEMI Network. This network is structured into: i) HUB centers – cardiology units with 24-hour hemodynamics lab (e.g., Perugia, Terni, Foligno); ii) SPOKE centers/antennas – hospitals with EDs that do not have 24-hour cardiologists or cath labs (the USL Umbria 1 hospitals).

Given that USL Umbria 1 hospitals are SPOKE/antenna centers, the protocol for diagnosed STEMI or STEMI equivalent is: i) immediate activation – start diagnostic-therapeutic pathway, activate the 118 transport service, and notify the HUB center;¹⁵ ii) reperfusion strategy – the goal is to achieve reperfusion within 120 minutes of the first medical contact;¹⁶ iii) primary percutaneous coronary intervention (pPCI) is the preferred reperfusion strategy; iv) fibrinolysis is an alternative only if the pPCI target time cannot be achieved (110-120 minutes); v) transport preparation – actions include securing a 2nd IV access (left antecubital vein, 18G), applying defibrillator pads, preparing transport medications, and performing bilateral groin/forearm shaving.¹⁷

Delays to pPCI, such as non-timely ECG, transfer to a local Critical Care Unit (CCU) for diagnosis, or waiting for the cardiologist before activating transport procedures, are explicitly forbidden.¹⁸

Management of patients with a diagnosis of non-ST elevation myocardial infarction/unstable angina

When a diagnosis of STEMI has been excluded based on clinical presentation, patient history, and the initial ECG, the patient with suspected ACS is managed according to the NSTEMI ACS (NSTEMI/unstable angina) pathway.¹⁸

The HEART score is a validated tool used in the ED to estimate the 30-day risk of major adverse cardiovascular events (MACE), including myocardial infarction, need for revascularization, or death, in patients presenting with chest pain suggestive of ACS (Tables 1 and 2).¹⁹

Differential diagnosis for chest pain

The initial assessment must consider other life-threatening conditions.²⁰ Table 3 shows the most frequent differential diagnoses in patients admitted to the ED for chest pain.

Management of non-acute coronary syndrome chest pain

For all causes of chest pain not immediately suspected of being ACS (based on CPS and HEART Score), where ACS has been excluded or is considered less probable than other etiologies, the ED physician will evaluate whether to proceed with specific diagnostic pathways/tests based on clinical suspicion. Among the main causes of non-traumatic non-cardiac chest pain, it is important to highlight PE and aortic dissection.

Pulmonary embolism

PE represents the final effect of the sudden obstruction of the pulmonary artery or one of its branches by an embolus. This embolus forms in the deep venous circulation of the lower limbs or pelvis in some cases, and in the venous circulation drained by the superior vena cava in other cases, before migrating into the pulmonary arterial circulation *via* the lesser circulation.

PE can occur as a single event or result from multiple subsequent episodes; in the latter case, repeated, peripheral embolization of small thrombi (pulmonary microembolism) is the most frequent occurrence.

PE develops days after the onset of deep vein thrombosis and, in some cases, it can be fatal within 1 hour of symptom onset, particularly when not clinically suspected. In some patients, PE is accompanied by shock or hypotension and in many cases, patients present without shock but exhibit signs of right ventricular dysfunction or myocardial injury in laboratory tests, which suggest an unfavorable prognosis. The diagnostic process for PE is primarily driven by a high degree of clinical suspicion, which arises from integrating patient history, the presence of venous thromboembolism risk factors, clinical objective findings, and the results of first-level instrumental exams [12-lead electrocardiogram, chest X-ray, arterial blood gas analysis (ABG)].²¹

Once clinical suspicion is established, the pre-test clinical probability of PE is estimated using validated scoring systems (such as the Wells Score or the Revised Geneva Score).

In case of suspicion: perform rapid tests such as ECG, eventual chest X-ray, ABG, and blood tests (complete blood count, coagulation panel, D-dimer, renal and hepatic function, troponin and brain natriuretic peptide), if not previously performed.

PE can be categorized into two classes:²²

- high-risk PE: patients with suspected PE presenting with conditions of hemodynamic instability: shock or hypotension (or reduction for at least 15 minutes, not caused by a new-onset arrhythmia, hypovolemia, or sepsis). In these patients, adequate hemodynamic and respiratory support (O₂ therapy, cautious volume expansion, use of inotropic/vasopressor drugs) must be immediately ensured.
- non-high-risk PE: all patients with suspected PE who are hemodynamically stable.

Patients with clinical suspicion of PE in stable clinical conditions should proceed, as the first step of the diagnostic phase, with the evaluation of pre-test clinical probability. This evaluation is carried out based on clinical judgment and the use of scores (Table 4) in order to classify patients into probability categories corresponding to an increased prevalence of PE. The two-level Wells Score is utilized.

- Patients with improbable PE (Wells 0-4): D-dimer testing should be performed. If negative, the diagnosis of PE can be ruled out; if positive, pulmonary computed tomography angiography (CTPA) must be performed. D-dimer testing may not be indicative in patients already hospitalised, as numerous pathologies can cause an increase (e.g., malignancies, fractures, surgical interventions); however, it maintains its negative predictive value.
- Patients with probable PE (Wells \$>4\$): In the absence of contraindications, perform CTPA directly. If PE is radiologically confirmed, it is useful to extend the computed tomography evaluation to the large abdominopelvic venous vessels to assess for potential involvement and the characteristics of the thrombotic formations.

Acute aortic syndrome

Acute aortic syndromes include aortic dissection, intramural hematoma, penetrating aortic ulcer, and aortic trauma.

The “acute” clinical presentation of AAS is often the first sign of the disease, and the rapidity of diagnosis and identification of the most appropriate therapeutic strategy is necessary to reduce an extremely unfavorable prognosis. This aspect differentiates them from other aortic pathologies that may have a delayed diagnosis, even after a long period of subclinical development.

Acute aortic dissection (AAD) is clinically suspected in less than half of the people in whom it is subsequently diagnosed. Although many confounding clinical factors delay an early and accurate diagnosis, the primary one is the “signal-to-noise” ratio.²³

AAD, which represents the most frequent pathology within AAS, clinically presents with acute, penetrating chest pain that may radiate posteriorly towards the back, following the extent of the pathology (migratory). It may be associated with symptoms of organ ischemia, such as stroke, mesenteric, renal, or lower limb ischemia, and ACS. Clinical objective findings may be normal or

may present signs of poor organ perfusion (neurological signs, absent/weak distal pulses, oligoanuria) or external rupture (cardiac tamponade, aortic valve insufficiency, hemothorax, hemoperitoneum).

The following table proposes the stratification of patients with suspected Aortic Dissection into three categories (low, intermediate, and high risk) based on the presence/absence of risk factors (Table 5).

Conclusions

The standardized protocol for managing non-traumatic chest pain in the USL Umbria 1 EDs represents a cohesive, multi-disciplinary approach aligned with international guidelines and the regional STEMI network.²⁰ By emphasizing rapid ECG, risk scoring (CPS and HEART), and streamlined transfer logistics for STEMI, the procedure ensures that critical life-saving interventions are delivered with optimal speed, ultimately contributing to reduced mortality and improved outcomes for patients presenting with suspected ACS.¹⁷

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Table 1. The HEART Score for chest pain risk stratification.

Component	0 points	1 point	2 points
History	Non-specific pain, not suggestive of ACS	Moderately suspicious for ACS	Highly suspicious for ACS
ECG	Normal	Non-specific repolarization changes (ST depression <0.5 MM or T-wave inversion)	Significant ST-segment depression >0.5 mm or transient ST elevation
Age	<65 years	65-89 years	≥90 years
Risk factors*	No known risk factors	1-2 known risk factors	≥3 known risk factors or history of CAD
Troponin	Normal/below the limit of detection	ULN	>3 X ULN

ACS, acute coronary syndrome; ULN, upper limit of normal; CAD, coronary artery disease *Risk factors include hypertension, hypercholesterolemia, diabetes mellitus, smoking, and family history of CAD.

Table 2. 30-day risk of major adverse cardiovascular events.

High	MACE probability >50%
Intermediate	MACE probability >12% and <17%
Low	MACE probability <2%

MACE, major adverse cardiovascular events

Table 3. Most frequent differential diagnoses for chest pain.¹⁹

Condition	Key clinical features
Acute coronary syndrome	Sweating, tachypnea, tachycardia, hypotension; exam may be normal in uncomplicated cases.
Pulmonary embolism	Tachycardia and dyspnea in Sat >90% of patients.
Aortic dissection	Severe, sudden onset pain; pulse deficit in limbs (30%); widened mediastinum on chest X-ray ⁵⁴ . Syncope in 10% of cases.
Oesophageal rupture	Emesis, subcutaneous emphysema, pneumothorax.
Pericarditis	Fever, pleuritic chest pain, exacerbated in the supine position (clinostatism), pericardial friction rub.
Pneumothorax	Dyspnea and pain upon inspiration, unilateral diminished or absent breath sounds.
Musculoskeletal	Costochondritis, Tietze syndrome (pain in costochondral joints).

Table 4. Wells Score.²³

Clinical characteristics	Score
Clinical signs and symptoms of DVT (minimum leg swelling and tenderness along the deep veins)	3
Alternative diagnosis less likely than PE	3
Heart rate > 100\$ beats/min	1.5
Immobilization for a period of >3 days or surgery in the preceding 4 weeks	1.5
Previous DVT or PE episode	1.5
Hemoptysis	1
Malignancy (ongoing treatment or within the last 6 months or palliative care)	1
Total Score > 4: PE probable	
Total Score < 4: PE improbable	

DVT, deep vein thrombosis; PE, pulmonary embolism.

Table 5. Aortic dissection Detection Risk Score.²⁴

Predisposing conditions	Pain characteristics	Clinical signs
Marfan syndrome/connective tissue disorders	Onset at its peak intensity within minutes	Perfusion deficit (pulse deficit/focal neurological deficit in the presence of pain/difference in limbs)
Family history	Severe intensity	Aortic insufficiency murmur (new or previously unknown if pre-existing) in the presence of pain
Known aortic valve disease	Tearing, stabbing, or ripping quality	Shock, hypotension
Recent cardiac surgery or catheterisation with aortic manipulation		
Known thoracic aortic aneurysm		
Low risk = no item, score 0	Intermediate risk = item from only one category, score 1	High risk = items belonging to 2 or 3 risk categories, score 2-3