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A Non–ST-Segment Elevation Myocardial Infarction: An Emergent Condition-Main Role of Emergency Providers and Clinical Pathologists: A Review

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Abstract:

Background: Non-ST-segment elevation myocardial infarction (NSTEMI) is a critical subtype of acute coronary syndrome (ACS) characterized by partial coronary occlusion and elevated cardiac biomarkers. Despite lacking ST elevation on ECG, NSTEMI carries significant morbidity and mortality risks, necessitating prompt diagnosis and management. Emergency providers and clinical pathologists play pivotal roles in early recognition, diagnosis, and intervention.

Aim: This review examines the pathophysiology, diagnostic challenges, and evidence-based management strategies for NSTEMI, emphasizing the collaborative roles of emergency and laboratory medicine teams.

Methods: A synthesis of current guidelines and clinical studies was conducted, focusing on risk stratification (e.g., GRACE, TIMI scores), biomarker interpretation (high-sensitivity troponin), and therapeutic protocols (anticoagulation, revascularization timing).

Results: NSTEMI accounts for 70% of ACS cases, with outcomes heavily influenced by timely troponin testing and ECG interpretation. Key findings include that High-sensitivity troponin assays enable earlier diagnosis (within 2–4 hours) but require serial measurements. Early invasive strategies (<24 hours) reduce mortality in high-risk patients (GRACE score >140). Complications like heart failure (15–20% incidence) and renal dysfunction (25–30%) underscore the need for multidisciplinary care.

Conclusion: Effective NSTEMI management hinges on coordinated efforts between emergency providers (rapid triage), pathologists (biomarker accuracy), and cardiologists (definitive care). Future directions include refined risk assessment tools and personalized therapies.

Keywords: NSTEMI, acute coronary syndrome, troponin, emergency medicine, clinical pathology, risk stratification.

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Introduction:

Acute coronary syndrome (ACS) encompasses three primary subtypes: ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina. This condition is associated with high rates of illness and death, making rapid diagnosis and proper treatment critical. While the diagnosis and management of STEMI are covered in other sections, NSTEMI and unstable angina share many similarities, with the key distinction being the presence of elevated cardiac biomarkers in NSTEMI. The following discussion focuses on the clinical presentation, diagnostic approach, and treatment strategies for NSTEMI [1-3]. ACS represents a spectrum of conditions resulting from reduced blood flow to the heart muscle, often due to coronary artery blockage. Among these, STEMI is characterized by complete arterial occlusion, whereas NSTEMI and unstable angina typically involve partial blockages. Unstable angina differs from NSTEMI in that it does not lead to detectable myocardial necrosis, as reflected by normal cardiac biomarker levels. Both conditions, however, require urgent evaluation to prevent further complications such as heart failure or arrhythmias. The management of NSTEMI involves risk stratification, anti-ischemic therapy, and often invasive strategies like coronary angiography and revascularization. Early intervention can significantly improve outcomes and reduce mortality rates [1-3]. Given the overlapping features of NSTEMI and unstable angina, clinicians must rely on biomarker testing and electrocardiographic findings to differentiate between them. Timely and accurate diagnosis ensures appropriate treatment, which may include medications like antiplatelets, anticoagulants, and statins, as well as lifestyle modifications. The following sections will explore the clinical manifestations, diagnostic criteria, and therapeutic approaches for NSTEMI in greater detail [1-3].

Etiology:

The underlying causes of non-ST-segment elevation myocardial infarction (NSTEMI) are diverse, with multiple contributing risk factors. Key etiological factors include tobacco use, sedentary lifestyle, hypertension, dyslipidemia (elevated cholesterol), diabetes mellitus, obesity, and a genetic predisposition due to a family history of cardiovascular disease. These risk factors contribute to the development of atherosclerosis, which leads to plaque formation and subsequent coronary artery narrowing or occlusion. In NSTEMI, partial or intermittent blockage of the coronary artery reduces blood flow, causing myocardial ischemia without complete vessel obstruction. Additional triggers may include inflammatory conditions, vasospasm, or embolic events, further compromising coronary blood flow. Identifying and addressing these risk factors is crucial in both prevention and management strategies for NSTEMI [1-4].

Epidemiology:

In the United States, the median age of patients presenting with acute coronary syndrome (ACS) is 68 years, with a notable gender disparity—males are affected more frequently than females in a 3:2 ratio. Annually, the U.S. records over 780,000 ACS cases, of which approximately 70% are classified as non-ST-segment elevation myocardial infarction (NSTEMI). This high prevalence underscores NSTEMI as the most common form of ACS, reflecting its significant burden on healthcare systems. Demographic variations exist, with older adults, men, and individuals with underlying cardiovascular risk factors being disproportionately affected. Additionally, socioeconomic factors, geographic location, and access to healthcare influence incidence and outcomes. The rising prevalence of metabolic disorders, such as diabetes and obesity, further contributes to the growing incidence of NSTEMI. Understanding these epidemiological trends is essential for targeted prevention strategies and resource allocation in cardiovascular care [5,6].

History and Physical

The classic presentation of non-ST-segment elevation myocardial infarction (NSTEMI) typically involves substernal chest pain described as pressure-like or squeezing in quality, which may occur at rest or with minimal exertion. This discomfort often persists for more than 10 minutes and frequently radiates to the jaw, neck, or either arm. Associated symptoms commonly include dyspnea, nausea, vomiting, diaphoresis, fatigue, or syncope. Notably, sudden-onset dyspnea without accompanying chest pain may also represent a significant presentation of acute coronary syndrome (ACS). Established risk factors for ACS include male gender, advanced age, family history of coronary artery disease (CAD), diabetes mellitus, pre-existing CAD, and renal insufficiency [7-9]. Atypical presentations of NSTEMI occur more frequently in specific patient populations, including women, individuals aged over 75 years, and those with diabetes, renal impairment, or dementia. These atypical manifestations may include epigastric pain, stabbing or pleuritic chest discomfort, indigestion-like symptoms, or isolated dyspnea. While typical symptoms remain more common across all ACS presentations, clinicians should maintain a high index of suspicion for these alternative symptom patterns in high-risk groups [7-9]. The physical examination in suspected NSTEMI cases often yields nonspecific findings. Certain examination features, such as back pain (suggesting aortic dissection) or a pericardial friction rub (indicating pericarditis), may point toward alternative diagnoses for chest pain. However, no single physical finding definitively confirms ACS. The presence of signs consistent with heart failure (e.g., pulmonary rales, peripheral edema, or elevated jugular venous pressure) should heighten clinical concern for ACS, though these findings remain nonspecific. A comprehensive evaluation incorporating both history and physical examination remains essential for appropriate risk stratification and diagnostic accuracy in suspected NSTEMI cases [7-9].

Evaluation, Diagnosis, and Role of Clinical Pathologists of NSTEMI

The evaluation of suspected non-ST-segment elevation myocardial infarction (NSTEMI) relies on three key components: clinical history, electrocardiogram (ECG), and cardiac biomarkers. An ECG should be obtained immediately in any patient presenting with chest pain or symptoms concerning acute coronary syndrome (ACS). While a normal ECG does not exclude ACS or NSTEMI, specific patterns provide critical diagnostic information. ST-segment elevation or anterior ST depression should raise immediate concern for STEMI and prompt emergent reperfusion therapy unless proven otherwise. Findings more suggestive of NSTEMI include transient ST elevation, ST depression (particularly in multiple leads), or new T-wave inversions. Given the dynamic nature of ischemic changes, serial ECGs should be performed at regular intervals (typically every 15-30 minutes initially) or with recurrence of symptoms [7-9].

Cardiac troponin has emerged as the biomarker of choice for myocardial injury due to its superior sensitivity and specificity compared to traditional markers like creatine kinase-MB. Contemporary troponin assays demonstrate elevation within 2-4 hours of symptom onset in most cases, while high-sensitivity troponin (hsTn) assays can detect myocardial injury even earlier. The timing and magnitude of troponin elevation correlate with infarct size—larger infarcts typically produce earlier and more pronounced troponin increases. Most patients with true myocardial ischemia will demonstrate troponin elevation within 6 hours of symptom onset, making serial measurements crucial. A negative troponin at 6 hours effectively rules out myocardial infarction in most cases. Interpretation requires attention to assay-specific characteristics: conventional troponin assays typically use the 99th percentile upper reference limit as the cutoff, with most healthy individuals having undetectable levels, while hsTn assays often

report detectable (but normal) levels in healthy individuals [7-9].

Risk stratification tools can supplement clinical judgment in evaluating ACS, though none have proven superior to experienced clinician assessment. Commonly used instruments include:

- The TIMI risk score (evaluating 7 clinical and ECG variables)
- The GRACE risk score (incorporating hemodynamic parameters and renal function)
- The HEART score (particularly useful in emergency settings, combining History, ECG, Age, Risk factors, and Troponin)
- Other validated tools like the Sanchis score, Vancouver rule, HEARTS3 score, and Hess prediction rule

The diagnosis of NSTEMI requires:

1. Clinical symptoms consistent with ACS (typical chest pain or equivalent symptoms)
2. Troponin elevation above the 99th percentile upper reference limit
3. Absence of persistent ST-segment elevation meeting STEMI criteria

The distinction between NSTEMI and unstable angina rests solely on the presence of detectable troponin elevation—both represent the same pathophysiological spectrum of ACS, with NSTEMI indicating actual myocardial necrosis. This differentiation has prognostic significance, as troponin-positive patients generally face higher short-term cardiovascular risk [7-9].

Comprehensive Management of NSTEMI: Evidence-Based Strategies

The management of non-ST-segment elevation myocardial infarction (NSTEMI) requires a systematic, multi-modal approach focused on reducing myocardial ischemia, preventing complications, and improving long-term outcomes. Initial stabilization measures should commence immediately upon suspicion of ACS, even before definitive diagnosis confirmation. Current guidelines emphasize oxygen therapy only for hypoxemic patients (SpO_2 <90%), as routine supplemental oxygen in normoxic patients may paradoxically increase coronary vascular resistance and myocardial injury through free radical generation [1,10,11]. Aspirin remains the cornerstone of antiplatelet therapy, with a chewable 324 mg non-enteric coated formulation preferred for rapid buccal absorption. For aspirin-allergic patients, prasugrel 60 mg loading dose provides an effective alternative, though its use requires careful consideration of bleeding risks [12,13]. Nitroglycerin administration follows a tiered approach: sublingual 0.4 mg every 5 minutes (maximum 3 doses) for persistent pain, transitioning to intravenous infusion (starting at 5-10 mcg/min) for refractory symptoms or concomitant hypertension/heart failure, with strict avoidance in patients using phosphodiesterase inhibitors (within 24-48 hours) or presenting with right ventricular infarction due to the risk of catastrophic hypotension [10,11].

Risk Stratification and Diagnostic Confirmation

For patients with suspected ACS but inconclusive initial findings (non-diagnostic ECG and negative troponins), current protocols mandate serial biomarker assessment (high-sensitivity troponin at 0, 3, and 6 hours) coupled with repeat ECGs during symptomatic episodes. This monitoring occurs in dedicated chest pain units or observation departments, where approximately 60-70% of patients can be safely discharged after rule-out protocols. Intermediate-risk patients often undergo functional testing before discharge, with exercise stress echocardiography or myocardial perfusion imaging preferred for their superior sensitivity (85-90%) compared to standard treadmill tests. Advanced centers increasingly employ coronary CT angiography as a gatekeeper to invasive strategies,

particularly in low-to-intermediate risk patients, demonstrating 95% negative predictive value for excluding significant CAD [1,13]. The emergence of accelerated diagnostic pathways (ADPs) incorporating the HEART score with 0- and 2-hour hsTn measurements has reduced unnecessary hospitalizations while maintaining patient safety, with validation studies showing >99% sensitivity for major adverse cardiac events at 30 days when combined with clinical judgment [11,13].

Anticoagulation and Invasive Strategy Considerations

Once the NSTEMI diagnosis is confirmed, immediate anticoagulation forms the therapeutic foundation. Current ACC/AHA guidelines recommend individualized selection from four approved options: unfractionated heparin (UFH) (60 IU/kg bolus, 12 IU/kg/hr infusion), enoxaparin (1 mg/kg subcutaneous every 12 hours), bivalirudin (0.1 mg/kg bolus, 0.25 mg/kg/hr infusion), or fondaparinux (2.5 mg subcutaneous daily). UFH remains prevalent in catheterization labs due to its rapid reversibility, while fondaparinux demonstrates superior safety in conservative management but requires UFH supplementation during PCI. The critical decision point involves the timing of invasive strategy: early angiography (<24 hours) benefits high-risk patients (GRACE score >140, recurrent ischemia, hemodynamic instability), reducing infarct progression by 38% compared to delayed approaches. Conversely, stabilized low-risk patients may benefit from ischemia-guided medical therapy, particularly with extensive comorbidities [1,10,12].

Pharmacological Management and Secondary Prevention

Admission to coronary care units facilitates intensive monitoring and initiation of evidence-based therapies. Beta-blockers (e.g., metoprolol tartrate 25-50 mg every 6-12 hours) should commence within 24 hours unless contraindicated (heart failure with reduced ejection fraction <40%, heart block >1st degree, or bronchospasm), reducing myocardial oxygen demand and arrhythmia risk. RAAS inhibition with ACE inhibitors (e.g., ramipril 2.5-5 mg daily) proves particularly beneficial in patients with LV dysfunction (EF<40%), diabetes, or hypertension, demonstrating 20% mortality reduction at 1 year. High-intensity statin therapy (atorvastatin 40-80 mg or rosuvastatin 20-40 mg) provides pleiotropic benefits beyond lipid-lowering, including plaque stabilization and reduced inflammatory markers. Contemporary NSTEMI management also emphasizes:

- Dual antiplatelet therapy (DAPT) with P2Y12 inhibitors (clopidogrel 600 mg load/75 mg daily, ticagrelor 180 mg load/90 mg BID, or prasugrel 60 mg load/10 mg daily for PCI patients) for 12 months unless bleeding risk prevails
- Consideration of aldosterone antagonists in post-MI patients with EF≤40% and either diabetes or heart failure
- Comprehensive cardiac rehabilitation referrals, shown to improve functional capacity by 15-30% and reduce readmissions [1,10,12,13].

Special Considerations and Emerging Therapies

Management nuances require attention to specific populations. Elderly patients (>75 years) warrant careful bleeding risk assessment (CRUSADE score) and often require reduced anticoagulant dosing. Diabetic patients benefit from more aggressive glycemic control (target HbA1c <7%) but should avoid hypoglycemia-induced ischemia. Recent trials investigating complete revascularization in multivessel disease (COMPLETE trial) support addressing non-culprit lesions during index hospitalization, reducing cardiovascular death by 26%. Novel therapies like low-dose rivaroxaban (2.5 mg BID) added to DAPT in high-risk patients (ATLAS ACS 2-TIMI 51) show promise but require careful bleeding monitoring. The evolving landscape of NSTEMI management continues to emphasize personalized medicine approaches, integrating

genomic profiling (CYP2C19 testing for clopidogrel metabolism) and advanced imaging to optimize outcomes [1,11,13].

Differential Diagnosis of NSTEMI

The evaluation of patients presenting with chest pain, nonspecific ECG changes, and elevated troponin requires careful consideration of multiple clinical conditions that can mimic non-ST-segment elevation myocardial infarction (NSTEMI). Myocarditis frequently presents with troponin elevation due to direct myocardial injury from viral or autoimmune inflammation, often accompanied by diffuse ST/T-wave abnormalities rather than localized ischemic patterns. Pericarditis typically demonstrates diffuse ST elevations and PR depression on ECG, with troponin elevation occurring in 30-50% of cases due to epicardial inflammation. Pulmonary embolism may cause right heart strain, manifesting as anterior T-wave inversions and troponin release from acute right ventricular pressure overload, often accompanied by hypoxia and elevated D-dimer [14-16]. Cardiovascular conditions such as left ventricular aneurysm can produce persistent ST elevations and troponin elevation from chronic wall motion abnormalities. Prinzmetal (variant) angina causes transient coronary vasospasm leading to ST elevations and troponin leaks that typically resolve with vasodilator therapy. Non-cardiac conditions must also be considered - anxiety disorders can produce chest pain with hyperventilation-related ECG changes (e.g., ST depression in inferior leads), while severe aortic stenosis may cause demand ischemia with troponin elevation due to chronic pressure overload. Hypertensive emergencies can precipitate myocardial injury through supply-demand mismatch, often showing diffuse ECG changes without coronary occlusion [17,18].

Critical distinctions include the temporal pattern of troponin elevation (prolonged in NSTEMI versus transient in many mimics) and associated clinical features. Myocarditis often follows viral prodromes, pericarditis shows positional pain improvement, and pulmonary embolism demonstrates right heart strain signs. These differentials necessitate comprehensive evaluation including echocardiography, coronary angiography when indicated, and sometimes advanced imaging (CMR for myocarditis, CTPA for PE) to establish the correct diagnosis and guide appropriate management [14,15,17,18].

Prognosis in NSTEMI: Short-term and Long-term Outcomes

The prognosis of non-ST-segment elevation myocardial infarction (NSTEMI) demonstrates a paradoxical relationship with unstable angina (UA), where NSTEMI patients exhibit lower 6-month mortality rates (3-5%) compared to UA patients (5-8%), despite having definitive myocardial necrosis. This apparent contradiction stems from more aggressive treatment protocols typically initiated for troponin-positive patients, including earlier coronary intervention and intensified pharmacotherapy. However, long-term outcomes (1-5 years) often reverse this trend, with NSTEMI patients facing higher mortality rates due to greater underlying coronary disease burden and myocardial damage [14].

Key Prognostic Determinants:

1. **Biomarker Severity**: The magnitude of troponin elevation directly correlates with both short- and long-term mortality. Patients with peak high-sensitivity troponin T (hsTnT) >1,000 ng/L face 3-fold higher 30-day mortality compared to those with hsTnT 50-200 ng/L. This reflects the extent of myocardial injury and microvascular obstruction [14].

2. **Comorbid Disease Burden**:

- **Diabetes Mellitus**: Insulin-dependent diabetes increases 1-year mortality by 40% due to accelerated atherosclerosis and microvascular dysfunction.
- **Chronic Kidney Disease (CKD)**: Each 10 mL/min decrease in eGFR below 60

mL/min/1.73m² raises mortality risk by 18%, partly due to uremic platelet dysfunction and delayed drug clearance.

- **Cognitive Impairment**: Dementia independently predicts 2-fold higher mortality, often from treatment non-adherence and delayed symptom recognition [14].

3. **Vascular Disease Complexity**: Patients with multivessel coronary disease or concomitant peripheral artery disease (ankle-brachial index <0.9) experience 50% higher 5-year mortality rates. This underscores the systemic nature of atherosclerotic disease in NSTEMI prognosis [14].

Risk Stratification Tools:

The GRACE 2.0 score provides the most robust mortality prediction, incorporating eight variables (age, Killip class, systolic BP, heart rate, creatinine, cardiac arrest, ST deviation, and troponin). Patients with GRACE scores >140 have 30-day mortality rates exceeding 10%, necessitating intensive intervention. Contemporary management protocols leveraging these predictors have reduced 1-year mortality rates from 12% (pre-2010) to 6-8% in modern registries through personalized treatment escalation [14].

Complications of NSTEMI: Pathophysiology and Clinical Implications

Non-ST-segment elevation myocardial infarction (NSTEMI) primarily leads to complications stemming from systemic hypoperfusion and myocardial dysfunction rather than the mechanical complications more commonly associated with STEMI (e.g., ventricular rupture, papillary muscle rupture, or aneurysms). The pathophysiology of these complications arises from transient or prolonged myocardial ischemia, resulting in impaired cardiac output, neurohormonal activation, and end-organ dysfunction [19].

1. Cardiac Complications

- **Left Ventricular Dysfunction (LVD):** NSTEMI can induce diffuse myocardial stunning or hibernation, leading to transient or persistent systolic dysfunction. Unlike STEMI, which often causes focal wall motion abnormalities, NSTEMI typically results in global hypokinesis due to microvascular dysfunction and repetitive ischemic insults. Severe cases may progress to ischemic cardiomyopathy, particularly in patients with pre-existing coronary artery disease [19].

- **Pulmonary Edema:** Acute decompensated heart failure occurs in 15-20% of NSTEMI patients, primarily due to elevated left ventricular filling pressures. This complication is more common in elderly patients, those with prior myocardial infarction, or individuals with significant multivessel disease.

- **Arrhythmias:** Atrial fibrillation (AF) occurs in ~10% of cases due to atrial ischemia and elevated left atrial pressures. Ventricular arrhythmias (e.g., non-sustained VT) may also develop but are less frequent than in STEMI [19].

2. Systemic and End-Organ Complications

- **Renal Dysfunction:** Approximately 25-30% of NSTEMI patients develop acute kidney injury (AKI) due to reduced renal perfusion, contrast exposure during angiography, or neurohormonal activation (e.g., RAAS overactivity). AKI significantly worsens prognosis, increasing in-hospital mortality by 3-5 fold [19].

- **Thromboembolic Events:** Systemic embolization from left ventricular thrombus is rare in NSTEMI compared to STEMI, but patients with severe LV dysfunction (EF <30%) remain at risk.

- **Recurrent Ischemia and Infarction:** Due to incomplete coronary occlusion, NSTEMI patients have a 5-10% risk of reinfarction within

30 days, particularly if revascularization is delayed or incomplete.

3. Long-Term Sequelae

- **Chronic Heart Failure (HF):** Up to 20% of survivors develop HF with reduced ejection fraction (HFrEF) within 1 year, especially if early revascularization is not performed.
- **Increased Mortality Risk:** While in-hospital mortality is lower than in STEMI (2-4% vs. 5-7%), long-term mortality converges at 1 year due to recurrent events and comorbidities [19].

Risk Mitigation Strategies

- Early invasive strategy (angiography within 24 hours) reduces reinfarction and HF rates.
- Guideline-directed medical therapy (GDMT), including beta-blockers, ACE inhibitors, and statins, improves outcomes.
- Close monitoring for AKI and hemodynamic instability is essential in high-risk patients. NSTEMI complications predominantly result from systemic hypoperfusion and myocardial dysfunction rather than structural damage. Prompt revascularization and aggressive medical management are crucial to mitigating adverse outcomes [19].

Deterrence and Patient Education in NSTEMI

Effective long-term management of non-ST-segment elevation myocardial infarction (NSTEMI) requires comprehensive patient education and lifestyle modifications to prevent recurrent cardiovascular events. Medication adherence is a cornerstone of secondary prevention, as discontinuation of antiplatelet agents, statins, or beta-blockers significantly increases the risk of reinfarction and mortality. Patients should receive clear instructions on the importance of taking prescribed medications, particularly dual antiplatelet therapy (DAPT), which must be continued for the recommended duration unless contraindicated. Pharmacist-led counseling and pill organizers can improve compliance, especially in elderly patients or those on complex regimens. Smoking cessation is one of the most critical interventions, as continued tobacco use doubles the risk of recurrent myocardial infarction. Healthcare providers should offer structured smoking cessation programs, including behavioral therapy and pharmacologic aids such as nicotine replacement therapy (NRT), varenicline, or bupropion. Even brief clinician advice increases quit rates, making it essential to address smoking at every follow-up visit. Dietary modifications, including a Mediterranean-style diet rich in fruits, vegetables, whole grains, and healthy fats, have been shown to reduce cardiovascular mortality. Patients should also be encouraged to engage in regular physical activity, ideally 150 minutes of moderate-intensity exercise per week, as supervised cardiac rehabilitation programs improve functional capacity and reduce readmission rates. Additionally, weight management and blood pressure control are vital, particularly in patients with comorbid diabetes or metabolic syndrome. Finally, stress management and mental health support should not be overlooked, as depression and anxiety are common post-NSTEMI and negatively impact recovery. A multidisciplinary approach involving cardiologists, primary care providers, dietitians, and mental health professionals ensures the best outcomes in secondary prevention [18,19].

Enhancing Healthcare Team Outcomes in NSTEMI Management

The optimal management of non-ST-segment elevation myocardial infarction (NSTEMI) requires a well-coordinated interprofessional team approach involving cardiologists, internists, emergency physicians, nurse practitioners, pharmacists, and cardiac rehabilitation specialists. This collaborative model ensures timely diagnosis, appropriate risk stratification, and evidence-based treatment, ultimately improving patient outcomes. Upon diagnosis or strong clinical suspicion of NSTEMI, immediate anticoagulation therapy should be initiated to prevent thrombus progression. The

choice of anticoagulant—whether unfractionated heparin (UFH), low-molecular-weight heparin (LMWH), bivalirudin, or fondaparinux—depends on institutional protocols and the likelihood of early percutaneous coronary intervention (PCI). Given the complexity of anticoagulation strategies, early cardiology consultation is crucial, particularly in high-risk patients where invasive management is anticipated. Pharmacists play a key role in dosing adjustments, monitoring bleeding complications, and ensuring drug interactions are minimized, especially in patients with renal impairment or those on multiple antithrombotic agents. Following diagnosis, patients should be admitted to a cardiac care unit for continuous monitoring. Beta-blockers should be initiated within 24 hours in the absence of contraindications (e.g., acute heart failure, bradycardia, or severe bronchospasm) to reduce myocardial oxygen demand and prevent arrhythmias [19].

ACE inhibitors (or ARBs if intolerant) are recommended for patients with left ventricular dysfunction (ejection fraction <40%), hypertension, diabetes, or chronic kidney disease, as they improve long-term survival and reduce adverse remodeling. High-intensity statin therapy is essential for lipid management and plaque stabilization, with pharmacists ensuring proper dosing and adherence. Risk stratification guides further management—high-risk patients (e.g., those with recurrent ischemia, hemodynamic instability, or significant troponin elevation) benefit from early invasive strategies (coronary angiography ± revascularization within 24 hours), whereas stable, low-risk patients may be managed conservatively with medical therapy and non-invasive testing. Nurses are instrumental in monitoring complications (e.g., bleeding, arrhythmias, or heart failure) and educating patients on medication adherence and lifestyle modifications. Long-term outcomes in NSTEMI depend on the extent of myocardial injury, adherence to secondary prevention measures, and control of comorbidities. Patients who fail to modify risk factors (e.g., smoking, poor diet, or sedentary lifestyle) face higher rates of recurrent events and mortality. A structured cardiac rehabilitation program, involving exercise training, dietary counseling, and psychosocial support, significantly enhances recovery and reduces readmissions. Regular follow-up with primary care providers ensures continuity of care, optimization of medications, and management of chronic conditions. By fostering seamless communication and role clarity among team members, this interprofessional approach minimizes errors, reduces delays in treatment, and improves both short- and long-term outcomes for NSTEMI patients [19].

Role of Emergency Providers:

The emergency provider plays a critical role in the initial recognition, risk stratification, and early management of NSTEMI, serving as the first point of contact for patients presenting with acute coronary symptoms. Upon patient arrival, emergency physicians must rapidly obtain a focused history and physical exam, with particular attention to characteristic ischemic chest pain features, risk factors for coronary artery disease, and potential alternative diagnoses. Immediate 12-lead ECG interpretation is essential to identify ST-segment deviations or T-wave abnormalities suggestive of ischemia, though a normal ECG does not exclude NSTEMI. Emergency providers should initiate first-line therapies including aspirin (324 mg chewable), sublingual nitroglycerin for ongoing pain, and supplemental oxygen only if hypoxemic (SpO2 <90%), while concurrently ordering serial high-sensitivity troponin measurements at presentation and 3–6-hour intervals to detect myocardial injury. Risk stratification using validated tools like the HEART or TIMI scores helps guide disposition decisions, with high-risk patients requiring urgent cardiology consultation and consideration for early invasive strategy. Emergency providers must also recognize and manage potential complications such as arrhythmias or acute heart failure while avoiding inappropriate fibrinolytic administration, which is

contraindicated in NSTEMI. Coordination with cardiology, pharmacy, and nursing staff is crucial to ensure timely anticoagulation initiation, appropriate pain control, and safe transition to inpatient care. The emergency department's efficiency in implementing these evidence-based protocols significantly impacts door-to-treatment times and subsequent clinical outcomes, making the emergency provider's role pivotal in the chain of survival for NSTEMI patients. Furthermore, emergency clinicians bear responsibility for patient education regarding the acute condition and the importance of follow-up care, particularly for those discharged after rule-out protocols, ensuring continuity in cardiovascular risk reduction strategies [20].

Conclusion:

NSTEMI represents a complex clinical entity where outcomes depend on seamless integration of diagnostic acuity and therapeutic precision. This review highlights several critical takeaways for optimizing patient care. First, the diagnostic paradigm for NSTEMI has evolved with high-sensitivity troponin assays, enabling detection of myocardial injury within hours of symptom onset. However, challenges persist in differentiating NSTEMI from mimics like myocarditis or pulmonary embolism, necessitating a combination of ECG findings, biomarker trends, and clinical context. The management of NSTEMI demands a risk-stratified approach. High-risk patients (e.g., those with hemodynamic instability or recurrent ischemia) benefit from early invasive coronary angiography, which reduces infarct progression by 38% compared to delayed strategies. Pharmacologically, dual antiplatelet therapy (DAPT) and anticoagulation form the cornerstone of treatment, though bleeding risks—particularly in elderly or renally impaired patients—require careful mitigation through tools like the CRUSADE score. Long-term outcomes are profoundly influenced by secondary prevention. Structured cardiac rehabilitation programs, which improve functional capacity by 15–30%, remain underutilized despite robust evidence. Similarly, adherence to guideline-directed medical therapy (e.g., statins, beta-blockers) is suboptimal in real-world practice, contributing to higher 1-year mortality rates (6–8%) despite advances in acute care. The emergency department serves as the critical gateway for NSTEMI management, where door-to-ECG and door-to-troponin times directly impact prognosis. Emergency providers must balance rapid rule-out protocols (e.g., HEART score) with judicious resource use, while clinical pathologists ensure accurate biomarker interpretation amid evolving assay technologies. Future directions include the integration of advanced imaging (e.g., coronary CTA for low-risk patients) and personalized approaches leveraging genetic testing (e.g., CYP2C19 for clopidogrel metabolism). Additionally, addressing disparities in care access, particularly among women, elderly patients, and those with comorbidities, remains imperative to improve population-level outcomes. In summary, NSTEMI management exemplifies the necessity of interprofessional collaboration, where emergency providers, pathologists, and cardiologists each play distinct yet interdependent roles. By combining rapid diagnostics, risk-adapted therapies, and rigorous secondary prevention, healthcare teams can mitigate the substantial burden of this prevalent ACS variant.

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References:

1. Gilutz H, Shindel S, Shoham-Vardi I. Adherence to NSTEMI Guidelines in the Emergency Department: Regression to Reality. Crit Pathw Cardiol. 2019 Mar;18(1):40-46.
2. Piątek Ł, Wilczek K, Janion-Sadowska A, Gierlotka M, Gąsior M, Sadowski M. Outcomes of a routine invasive strategy in elderly patients with non-ST-segment elevation myocardial

infarction from 2005 to 2014: results from the PL-ACS registry. Coron Artery Dis. 2019 Aug;30(5):326-331.

3. Manfredonia L, Lanza GA, Crudo F, Lamendola P, Graziani F, Villano A, Locorotondo G, Melita V, Mencarelli E, Pennestrì F, Lombardo A, De Vita A, Ravenna SE, Bisignani A, Crea F. Diagnostic role of echocardiography in patients admitted to the emergency room with suspect no-ST-segment elevation acute myocardial infarction. Eur Rev Med Pharmacol Sci. 2019 Jan;23(2):826-832.
4. Yameny, A. Diabetes Mellitus Overview 2024. *Journal of Bioscience and Applied Research*, 2024; 10(3): 641-645. doi: 10.21608/jbaar.2024.382794
5. Rupprecht HJ, Geeren M, Geeren M, Weilemann S, Schuster HP. [Acute coronary syndrome without ST-elevation (NSTE-ACS)]. Herz. 2019 Feb;44(1):10-15.
6. Kamińska J, Koper OM, Siedlecka-Czykier E, Matowicka-Karna J, Bychowski J, Kemona H. The utility of inflammation and platelet biomarkers in patients with acute coronary syndromes. Saudi J Biol Sci. 2018 Nov;25(7):1263-1271.
7. Sriha Belguith A, Beltaief K, Msolli MA, Bouida W, Abroug H, Ben Fredj M, Zemni I, Grissa MH, Boubaker H, ESCor Investigators group. Hsairi M, Nouira S., ESCorT Investigators group. Management of acute coronary syndrome in emergency departments: a cross sectional multicenter study (Tunisia). BMC Emerg Med. 2018 Dec 03;18(1):50.
8. Lemkes JS, Janssens GN, van der Hoeven NW, van de Ven PM, Marques KMJ, Nap A, van Leeuwen MAH, Appelman YEA, Knaapen P, Verouden NJW, Allaart CP, Brinckman SL, Saraber CE, Plomp KJ, Timmer JR, Kedhi E, Hermanides RS, Meuwissen M, Schaap J, van der Weerdt AP, van Rossum AC, Nijveldt R, van Royen N. Timing of revascularization in patients with transient ST-segment elevation myocardial infarction: a randomized clinical trial. Eur Heart J. 2019 Jan 14;40(3):283-291.
9. Arora S, Stouffer GA, Kucharska-Newton A, Vaduganathan M, Qamar A, Matsushita K, Kolte D, Reynolds HR, Bangalore S, Rosamond WD, Bhatt DL, Caughey MC. Fifteen-Year Trends in Management and Outcomes of Non-ST-Segment-Elevation Myocardial Infarction Among Black and White Patients: The ARIC Community Surveillance Study, 2000-2014. J Am Heart Assoc. 2018 Oct 02;7(19):e010203.
10. Puymirat E, Bonaca M, Fumery M, Tea V, Aissaoui N, Lemesles G, Bonello L, Ducrocq G, Cayla G, Ferrières J, Schiele F, Simon T, Danchin N., FAST-MI investigators. Atherothrombotic risk stratification after acute myocardial infarction: The Thrombolysis in Myocardial Infarction Risk Score for Secondary Prevention in the light of the French Registry of Acute ST Elevation or non-ST Elevation Myocardial Infarction registries. Clin Cardiol. 2019 Feb;42(2):227-234.
11. Güntekin Ü, Tosun V, Kilinç AY, Saydam G, Korucuk N, Bozdemir MN. ST segment elevation myocardial infarction (STEMI) patients are more likely to achieve lipid-lowering treatment goals: A retrospective analysis of patients presenting with first acute coronary syndromes. Medicine (Baltimore). 2018 Sep;97(39):e12225.
12. Sanchis J, Ariza-Solé A, Abu-Assi E, Alegre O, Alfonso F, Barrabés JA, Baz JA, Carol A, Díez Villanueva P, García Del Blanco B, Elízaga J, Fernandez E, García Del Egido A, García Picard J, Gómez Blázquez I, Gómez Hospital JA, Hernández-Antolín R, Llibre C, Marín F, Martí Sánchez D, Martín R, Martínez Sellés M, Miñana G, Morales Gallardo MJ, Núñez J, Pérez de Prado A, Pinar E, Sanmartín M, Sionis A, Villa A, Marrugat J, Bueno H. Invasive Versus Conservative Strategy in Frail Patients With NSTEMI: The MOSCA-FRAIL Clinical Trial

Study Design. Rev Esp Cardiol (Engl Ed). 2019 Feb;72(2):154-159.

13. Bønaa KH, Steigen T. Coronary angiography in non-ST-elevation acute myocardial infarction - whom and when? Tidsskr Nor Laegeforen. 2017 Nov 28;137(22)
14. Sanchis J, Núñez J, Bodí V, Núñez E, García-Alvarez A, Bonanad C, Regueiro A, Bosch X, Heras M, Sala J, Bielsa O, Llácer A. Influence of comorbid conditions on one-year outcomes in non-ST-segment elevation acute coronary syndrome. Mayo Clin Proc. 2011 Apr;86(4):291-6.
15. Elbadawi A, Elgendy IY, Mahmoud K, Barakat AF, Mentias A, Mohamed AH, Ogunbayo GO, Megaly M, Saad M, Omer MA, Paniagua D, Abbott JD, Jneid H. Temporal Trends and Outcomes of Mechanical Complications in Patients With Acute Myocardial Infarction. JACC Cardiovasc Interv. 2019 Sep 23;12(18):1825-1836.
16. Yameny, A. D-dimer levels in COVID-19 out-hospitalized patients in Egypt. *Journal of Medical and Life Science*, 2021; 3(1): 19-24. doi: 10.21608/jmals.2021.200216
17. Harrington DH, Stueben F, Lenahan CM. ST-Elevation Myocardial Infarction and Non-ST-Elevation Myocardial Infarction: Medical and Surgical Interventions. Crit Care Nurs Clin North Am. 2019 Mar;31(1):49-64.
18. Held EP, Henry TD. When small vessels become big problems! Microvascular dysfunction in NSTEMI. Catheter Cardiovasc Interv. 2018 Nov 15;92(6):1075-1076.
19. Basit, H., Malik, A., & Huecker, M. R. (2023). Non–ST-segment elevation myocardial infarction. In *StatPearls [Internet]*. StatPearls Publishing.
20. Pollack Jr, C. V., & Braunwald, E. (2008). 2007 update to the ACC/AHA guidelines for the management of patients with unstable angina and non–ST-segment elevation myocardial infarction: implications for emergency department practice. *Annals of emergency medicine*, *51*(5), 591-606.

حالة طارئة – الدور الرئيسي لمقدمي الرعاية في الطوارئ وأخصائي علم الأمراض السريري: احتشاء عضلة القلب غير المرتبط بارتفاع المقطع ST

الملخص:

الخلفية: احتشاء عضلة القلب غير المرتبط بارتفاع المقطع (NSTEMI) ST هو نوع خطير من المتلازمة التاجية الحادة(ACS) ، يتميز بانسداد جزئي في الشريان التاجي وارتفاع في مؤشرات القلب الحيوية. رغم غياب ارتفاع المقطع ST في تخطيط القلب، إلا أن NSTEMI يرتبط بمعدلات مرتفعة من الممراضة والوفيات، ما يتطلب تشخيصاً وعلاجاً عاجلين. يلعب مقدمو الرعاية في أقسام الطوارئ وأخصائيو علم الأمراض السريري دوراً محورياً في التعرف المبكر والتدخل السريع.

الهدف: يستعرض هذا البحث الفسيولوجيا المرضية، والتحديات التشخيصية، والاستراتيجيات العلاجية المبنية على الأدلة لحالاتNSTEMI ، مع التركيز على الأدوار التكاملية لفريقي الطوارئ والمختبرات.

المنهجية: تمت مراجعة الأدلة الإرشادية والدراسات السريرية الحديثة، مع التركيز على تصنيف الخطورة) مثل نقاط GRACE و(TIMI، وتفسير المؤشرات الحيوية (التروبونين عالي الحساسية)، والبروتوكولات العلاجية (مضادات التخثر، توقيت إعادة التوعية).

النتائج: يمثل NSTEMI حوالي 70% من حالات المتلازمة التاجية الحادة. وتعتمد النتائج بشكل كبير على توقيت فحص التروبونين وتفسير تخطيط القلب. أبرز النتائج تشمل :فحوصات التروبونين عالية الحساسية تتيح تشخيصاً مبكراً (خلال 2–4 ساعات) ولكن تتطلب قياسات متسلسلة الاستراتيجيات التدخلية المبكرة (أقل من 24 ساعة) تقلل الوفيات لدى المرضى مرتفعي الخطورة (درجة GRACE >140) المضاعفات مثل فشل القلب (15–20%) والقصور الكلوي (25–30%) تؤكد الحاجة إلى رعاية متعددة التخصصات

الاستنتاج: تعتمد إدارة NSTEMI الفعالة على التعاون بين مقدمي الرعاية في الطوارئ (الفرز السريع)، وأخصائي علم الأمراض (دقة المؤشرات الحيوية)، وأطباء القلب (الرعاية التخصصية). وتشمل التوجهات المستقبلية تحسين أدوات تقييم الخطورة وتطوير علاجات مخصصة.

الكلمات المفتاحية :المتلازمة التاجية الحادة، التروبونين، طب الطوارئ، علم الأمراض السريري، تصنيف الخطورة