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Panic Attack or Medical Emergency? The Role of Biomarkers and Point-of-Care Testing in Emergency Department Decision-Making

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Abstract

Background: Panic attacks commonly produce chest pain, dyspnea, palpitations, dizziness, tremor, paresthesia, and fear of dying—features that overlap with acute coronary syndrome, pulmonary embolism, arrhythmia, metabolic disease, toxic exposure, and other time-critical disorders. Because panic attack has no confirmatory biomarker, emergency clinicians must first estimate and appropriately investigate organic risk without converting every low-risk presentation into indiscriminate testing. Objective: To critically review how biomarkers and point-of-care testing (POCT) can support the differentiation of suspected panic attacks from acute medical emergencies and to propose a practical, risk-adapted diagnostic framework. Methods: A narrative review was conducted using targeted searches of PubMed/MEDLINE and major professional-society guidance through August 2026. Search concepts included panic attack, panic disorder, emergency department, chest pain, dyspnea, high-sensitivity cardiac troponin, D-dimer, blood gas analysis, lactate, glucose, electrolytes, thyroid testing, electrocardiography, and point-of-care ultrasonography. Priority was given to guidelines, systematic reviews, prospective diagnostic studies, and literature directly relevant to emergency decision-making. Results: No laboratory or bedside test establishes panic attack. High-sensitivity cardiac troponin pathways, used with electrocardiography and structured clinical risk assessment, can rapidly exclude myocardial infarction in eligible patients. D-dimer is useful only after assessment of pretest probability for pulmonary embolism; age-adjusted or probability-adjusted thresholds reduce unnecessary imaging. Capillary glucose is a rapid test for an immediately reversible mimic. Blood gas analysis can document hypocapnia and respiratory alkalosis, but these findings are neither specific nor sufficient to exclude serious disease; lactate may rise during psychogenic hyperventilation. Electrolyte, thyroid, toxicologic, and inflammatory testing should be hypothesis driven. Point-of-care ultrasonography can rapidly identify alternative cardiopulmonary pathology but cannot, in isolation, rule out acute coronary syndrome or pulmonary embolism. Conclusions: The safest strategy is selective exclusion rather than a fixed 'panic panel.' Biomarkers and POCT should be chosen according to symptoms, red flags, pretest probability, test timing, and the consequences of a missed diagnosis. Reassessment after targeted testing is essential before a panic-related diagnosis and discharge plan are considered.

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Introduction

A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes and is accompanied by characteristic cognitive and somatic symptoms. It is a clinical event rather than a diagnosis confined to panic disorder: attacks may occur in other psychiatric disorders, in medical disease, or in association with substances. Current diagnostic criteria also require that symptoms are not better attributable to a substance or another medical condition [1]. This exclusion is particularly important in the emergency department (ED), where the same symptom cluster may be the first manifestation of a time-sensitive illness.

Chest pain, dyspnea, palpitations, tachycardia, sweating, tremor, dizziness, paresthesia, derealization, and fear of dying can accompany both panic and acute cardiopulmonary disease. Panic disorder is common in patients assessed for non-cardiac chest pain, but psychiatric comorbidity does not protect against organic disease [2-4]. Conversely, repeated low-yield investigations can reinforce health anxiety, prolong length of stay, expose patients to radiation and contrast, and fragment follow-up. The central task is therefore not to prove panic with a test, but to reduce the probability of relevant acute disease to an acceptable level using a calibrated combination of history, examination, electrocardiography (ECG), biomarkers, and bedside imaging.

The diagnostic value of a biomarker depends on pretest probability, timing, analytical characteristics, and the clinical action attached to the result. A normal test is not automatically a rule-out test, and an abnormal test is not automatically evidence of the feared diagnosis. High-sensitivity cardiac troponin (hs-cTn), D-dimer, glucose, blood gas variables, lactate, electrolytes, thyroid tests, and inflammatory markers each answer different clinical questions. Point-of-care ultrasonography (POCUS) similarly extends the physical examination but remains operator- and context-dependent.

This review evaluates the role and limitations of commonly available biomarkers and POCT in adults presenting with symptoms compatible with panic attack. It also proposes the PANIC-SAFE framework, intended as a cognitive aid for targeted exclusion, reassessment, and safe disposition rather than as a validated clinical decision rule.

Materials and Methods

A targeted narrative literature review was undertaken. PubMed/MEDLINE and professional-society guideline repositories were searched for English-language publications available through 31 August 2026. Search terms and combinations included: 'panic attack', 'panic disorder', 'anxiety', 'emergency department', 'chest pain', 'dyspnea', 'biomarker', 'point-of-care testing', 'high-sensitivity troponin', 'D-dimer', 'blood gas', 'hypocapnia', 'lactate', 'glucose', 'electrolytes', 'thyroid', 'electrocardiography', and 'point-of-care ultrasound'. Reference lists of key guidelines and reviews were screened for additional relevant studies.

Priority was assigned to international clinical guidelines, systematic reviews, prospective diagnostic studies, and studies addressing tests used during acute ED assessment. Evidence was synthesized around clinical questions rather than pooled quantitatively because of heterogeneity in populations, index tests, thresholds, outcomes, and the limited number of studies enrolling patients with a final diagnosis of panic attack. This article is therefore a narrative review and does not claim systematic-review completeness.

Results

The Diagnostic Starting Point: Pretest Probability

and Red Flags

Testing should follow, not replace, an initial clinical estimate. Immediate threats are suggested by hemodynamic instability, hypoxemia, syncope during exertion, persistent or exertional chest pressure, focal neurological deficit, new ischemic ECG changes, sustained arrhythmia, unilateral leg swelling, hemoptysis, recent surgery or immobilization, pregnancy/postpartum status, stimulant exposure, fever, major metabolic risk, or an atypical first episode at older age. A sudden sensation of fear does not establish psychiatric causation: autonomic activation is a normal response to hypoxemia, tachyarrhythmia, hypoglycemia, and shock.

The symptom trajectory is informative but not definitive. Panic symptoms typically peak rapidly and may improve with reassurance and normalization of ventilation. However, spontaneous improvement also occurs in paroxysmal supraventricular tachycardia, transient ischemia, and intermittent toxic or endocrine states. Diagnostic closure should therefore occur only after the clinician has reconciled symptoms, vital signs, examination, ECG, risk factors, and the results of appropriately selected tests.

Electrocardiography and Cardiac Biomarkers

A 12-lead ECG is the first-line bedside test when panic-like symptoms include chest pain, palpitations, syncope or near-syncope, unexplained dyspnea, or relevant cardiovascular risk. It can reveal acute ischemia, pre-excitation, Brugada pattern, QT prolongation, atrial fibrillation, supraventricular tachycardia, or ventricular arrhythmia. A normal ECG lowers concern for some conditions but does not exclude non-ST-elevation myocardial infarction (NSTEMI), intermittent arrhythmia, or pulmonary embolism.

Cardiac troponin is a marker of myocardial injury, not a test for anxiety and not, by itself, a diagnosis of myocardial infarction. Contemporary guidelines recommend hs-cTn assays within validated accelerated diagnostic pathways, interpreted as assay-specific concentrations and changes over time together with clinical assessment and ECG findings [5-7]. In eligible low-risk patients, rule-out algorithms can achieve a very high negative predictive value for myocardial infarction, but performance depends on time from symptom onset, assay, population, and protocol adherence [8-10]. Very early presenters may require repeat sampling. Conventional or point-of-care assays should not be inserted into hs-cTn algorithms unless specifically validated.

An elevated troponin should prevent a reflex label of panic but does not automatically mean type 1 myocardial infarction. Tachyarrhythmia, myocarditis, pulmonary embolism, heart failure, renal dysfunction, sepsis, strenuous exertion, and stress cardiomyopathy can cause myocardial injury [11]. Panic and catecholaminergic stress may coexist with these conditions. The clinical question shifts from 'Is this anxiety?' to 'What explains the injury, and is there acute ischemia?'

Risk scores such as HEART may support disposition in undifferentiated chest pain when used in an appropriate pathway, but scores do not supersede clinician judgment or serial ECG/troponin interpretation [6,12]. In a young patient with brief recurrent symptoms, a normal ECG and very low ischemic probability, troponin testing may be unnecessary; indiscriminate testing can produce incidental elevations and downstream cascades.

D-dimer and Suspected Pulmonary Embolism

Pulmonary embolism (PE) and panic attack share dyspnea, chest discomfort, tachycardia, dizziness, hyperventilation, and apprehension. D-dimer has high sensitivity but limited specificity and is useful only after estimating PE pretest probability. In patients at very low risk who satisfy all Pulmonary Embolism Rule-out Criteria (PERC), testing may be avoided; in low or intermediate probability, a negative high-sensitivity D-dimer can exclude PE without imaging [13-16]. Patients with high clinical probability should generally proceed directly to imaging rather than rely on D-dimer.

Age-adjusted D-dimer thresholds (age multiplied by 10 micrograms/L fibrinogen-equivalent units in patients aged 50 years or older, when the assay reports those units) and clinical-probability-adjusted strategies such as YEARS can reduce computed tomography pulmonary angiography while maintaining safety in validated populations [17,18]. These strategies must be used as complete protocols; thresholds and units are not interchangeable across assays.

A positive D-dimer does not distinguish PE from infection, inflammation, cancer, pregnancy, trauma, aging, or many other states. Ordering it in a patient with negligible pretest probability can create avoidable imaging. Conversely, calling marked dyspnea 'panic' without structured PE risk assessment is unsafe, particularly when tachycardia, hypoxemia, pleuritic pain, hemoptysis, venous thromboembolism history, estrogen exposure, or recent immobilization is present.

Blood gas Analysis, Hypocapnia, Lactate, and Electrolytes

Hyperventilation can produce hypocapnia, acute respiratory alkalosis, cerebral vasoconstriction, ionized hypocalcemia, paresthesia, carpopedal spasm, dizziness, and chest discomfort. Venous blood gas analysis is often sufficient for rapid assessment of pH and carbon dioxide trends in a stable patient, whereas arterial sampling is reserved for questions requiring accurate arterial oxygenation or when the clinical situation demands it [19,20]. A low PCO2 supports hyperventilation but is not specific for panic: PE, asthma, pneumonia, sepsis, pregnancy, hepatic failure, pain, and salicylate toxicity may produce the same pattern.

Lactate deserves careful interpretation. Hyperlactatemia is commonly associated with tissue hypoperfusion and severe illness, but it can also arise from adrenergic stimulation, impaired clearance, drugs, seizures, strenuous muscle activity, and respiratory alkalosis. Studies of psychogenic hyperventilation and panic-related attacks demonstrate that lactate may be elevated despite alkalemia and clinical stability [21,22]. Therefore, a modestly elevated lactate with marked hypocapnia is not proof of sepsis or shock; equally, it should not be dismissed until perfusion, vital signs, infection risk, toxic exposure, seizure, and other causes have been assessed. Serial clinical evaluation and lactate trends may be more informative than a single value.

Electrolyte testing is appropriate when weakness, tetany, prolonged QT, vomiting, diuretic use, malnutrition, renal disease, intoxication, or a severe/prolonged episode is present. Respiratory alkalosis decreases ionized calcium without necessarily reducing total calcium and may shift phosphate and potassium intracellularly. Severe hypophosphatemia associated with hyperventilation is uncommon but clinically relevant [23]. Magnesium, calcium, phosphate, and potassium abnormalities can both mimic anxiety and increase arrhythmic risk.

Glucose and Metabolic Testing

Capillary blood glucose is one of the highest-yield immediate POCTs in patients with altered behavior, diaphoresis, tremor, palpitations, weakness, dizziness, or confusion. Neurogenic hypoglycemia can closely reproduce a panic attack, while neuroglycopenia may cause cognitive change, seizure, or coma. A low value requires prompt treatment and evaluation of its cause; symptom resolution after glucose supports hypoglycemia but does not eliminate the need to identify medication-related, endocrine, hepatic, or septic contributors [24].

Additional metabolic testing should be selected according to context. A basic metabolic panel may identify sodium disorders, renal dysfunction, and acid-base clues. Ketone measurement is indicated when diabetes, hyperglycemia, vomiting, starvation, or sodium-glucose cotransporter-2 inhibitor use raises concern for ketoacidosis, including euglycemic presentations. Routine broad metabolic panels in every typical recurrent panic presentation are unlikely to add value.

Thyroid, Toxicologic, and Inflammatory Testing

Thyrotoxicosis can cause anxiety, tremor, heat intolerance, weight loss, diarrhea, atrial fibrillation, and persistent tachycardia. Thyroid-stimulating hormone (TSH) is a suitable initial test when clinical features suggest thyroid dysfunction; free thyroxine and, when appropriate, triiodothyronine clarify an abnormal TSH or strong suspicion [25]. Thyroid testing is rarely an emergency rule-out test for a brief, otherwise typical recurrent attack. It becomes urgent when thyroid storm is considered, a clinical diagnosis characterized by severe systemic decompensation rather than anxiety alone.

Substances and medications are important mimics. Caffeine, nicotine, cannabis, cocaine, amphetamines, decongestants, beta-agonists, thyroid hormone, anticholinergics, and withdrawal from alcohol or sedative-hypnotics may produce panic-like autonomic symptoms. A careful exposure history, prescription review, and focused examination often

outperform an indiscriminate urine drug screen. Standard immunoassays have false positives, false negatives, variable detection windows, and limited ability to establish acute impairment [26]. Specific testing is appropriate when it changes immediate management—for example, acetaminophen and salicylate concentrations in a possible overdose, carboxyhemoglobin with exposure concern, or targeted confirmatory toxicology.

Complete blood count, C-reactive protein, or procalcitonin may support evaluation when infection, anemia, or systemic inflammation is clinically plausible. None differentiates panic from organic disease. Leukocytosis can reflect catecholamine-mediated demargination, and procalcitonin should not be used alone to start or withhold antibiotics in suspected sepsis [27]. Biomarker results must remain subordinate to the infection source assessment, physiology, and serial examination.

Point-of-Care Ultrasonography

POCUS can rapidly identify or increase suspicion for pneumothorax, pericardial effusion with tamponade physiology, left ventricular systolic dysfunction, pulmonary edema, right ventricular strain, pleural effusion, abdominal aortic aneurysm, and selected other causes of acute symptoms. Lung ultrasound protocols have high diagnostic utility in acute dyspnea when integrated with clinical assessment [28-31]. Focused cardiac and lung views may be particularly valuable when vital signs are abnormal, auscultation is equivocal, or the clinician needs a rapid explanation for dyspnea or hypotension.

Important limitations prevent POCUS from becoming a universal rule-out test. A normal focused examination does not exclude acute coronary syndrome. Absence of right ventricular dilation does not exclude PE, particularly smaller or hemodynamically stable emboli; right ventricular strain is also not specific for acute PE. Lung sliding reduces the likelihood of pneumothorax at the scanned location but is technique- and context-dependent. Image acquisition and interpretation vary with training, body habitus, disease stage, and equipment. POCUS should answer a defined question and be documented with its scope and limitations.

Integrated Interpretation: Avoid the Fixed "Panic Panel"

The evidence does not support a universal laboratory bundle for suspected panic attack. A fixed panel ignores base rates and creates predictable harms: false-positive D-dimer results, incidental troponin elevations, nonspecific inflammatory changes, unnecessary radiation, and prolonged observation. A safer approach begins with the chief symptom and the dangerous diagnoses that remain plausible after initial assessment.

For chest pain, the core may be ECG with an hs-cTn pathway when acute coronary syndrome risk is not negligible. For dyspnea, pulse oximetry, ECG, lung/cardiac POCUS, and structured PE assessment guide whether D-dimer, imaging, blood gas, or other testing is warranted. For palpitations, rhythm capture, electrolytes, medication/substance review, and ambulatory follow-up may be more useful than broad biomarkers. For diaphoresis, tremor, or cognitive change, immediate glucose is essential. Persistent unexplained tachycardia should trigger reconsideration of infection, PE, anemia, endocrine disease, drugs, withdrawal, and arrhythmia rather than escalating reassurance alone.

Table 1: Risk-Adapted Use of Biomarkers and POCT in Panic-Like Presentations.

Presentation / question	Preferred first steps	Conditional tests	Key limitation
Chest pain / ACS?	ECG; history; risk assessment	Validated hs-cTn pathway	Normal ECG alone does not exclude NSTEMI; troponin indicates injury, not cause.
Dyspnea / PE?	SpO2; ECG; PE pretest probability; lung/cardiac POCUS	PERC or D-dimer strategy; imaging when indicated	Positive D-dimer is non-specific; normal POCUS does not exclude PE.
Palpitations / arrhythmia?	ECG and rhythm capture; medication/ substance review	Electrolytes; hs-cTn if ischemia/injury plausible; ambulatory monitoring	Symptoms may stop before ECG; sinus tachycardia requires a cause.
Hyperventilation / metabolic mimic?	Vital signs; glucose; focused examination	VBG/ABG; lactate; Ca/ Mg/phosphate according to severity	Hypocapnia and lactate elevation are not specific for panic.
Endocrine, toxic, infectious cause?	Exposure history; temperature; examination	TSH/free T4, targeted toxicology, CBC/CRP/ procalcitonin as indicated	Broad screening produc- es nonspecific abnormalities and rarely proves etiology.

Discussion

The principal finding of this review is conceptual: biomarkers and POCT improve safety when they are used to answer targeted exclusion questions, not when they are treated as a biochemical signature of panic. Panic attack remains a clinical diagnosis that can be made only after the presentation has been adequately explained and important alternatives have been reduced to an acceptably low probability. The acceptable threshold varies with the severity of the missed condition, reliability of follow-up, patient characteristics, and test performance.

The highest-value tests are those linked to a validated pathway. Examples include serial hs-cTn with ECG and clinical risk assessment for suspected acute coronary syndrome, and PERC/D-dimer strategies for suspected PE. By contrast, isolated lactate, leukocyte count, or TSH values rarely settle the acute differential. Blood gas findings can explain symptoms but cannot establish etiology. POCUS accelerates recognition of several dangerous alternatives, yet negative findings must be interpreted within the sensitivity of the focused examination.

Premature psychogenic attribution is a major hazard. The presence of a psychiatric history may generate anchoring and diagnostic overshadowing. A prior panic diagnosis should inform probability but never invalidate new red flags. Equally, repeated indiscriminate investigation carries harms and may undermine patient confidence. The clinician should explain what has been assessed, what the tests do and do not show, why a dangerous diagnosis is unlikely, and what changes require urgent reassessment.

After medical stabilization and targeted exclusion, acute management should include a calm environment, clear explanation, coached slow breathing rather than paper-bag rebreathing, assessment for suicidality and substance use when indicated, and an individualized follow-up plan. Benzodiazepines may relieve severe acute symptoms in selected patients but carry risks of sedation, impaired driving, dependence, and interaction with alcohol or opioids; they should not substitute for diagnostic assessment or longitudinal care. Evidence-based long-term management of panic disorder centers on cognitive-behavioral therapy and appropriate pharmacotherapy [32,33].

The Proposed PANIC-SAFE Framework

PANIC-SAFE is proposed as a bedside cognitive framework, not a validated score. Its purpose is to make the sequence of reasoning explicit and to reduce both premature closure and indiscriminate testing.

P — Primary stabilization: airway, breathing, circulation, mental status, temperature, oxygen saturation, and immediate treatment of instability. A — Assess red flags and pretest probability: define the dominant syndrome and time-sensitive alternatives. N — Necessary targeted biomarkers: order only tests linked to a plausible diagnosis and an action threshold. I — Immediate POCT: ECG, glucose, rhythm monitoring, blood gas, pregnancy testing when relevant, and focused ultrasound according to the presentation. C — Clinical reassessment: repeat vital signs and examination; reconcile symptom evolution with all results and reconsider discordant findings. SAFE — Safe disposition, Advice, Follow-up, and Explicit return precautions: document why urgent disease is unlikely, assess psychiatric risk when indicated, arrange continuity, and specify warning symptoms. The framework intentionally prevents a 'negative tests equal panic' shortcut. A patient may have a non-life-threatening organic diagnosis, a substance effect, or an undifferentiated syndrome requiring observation. Conversely, a patient with a well-characterized recurrent attack, normal vital signs, a reassuring examination and ECG, no red flags, and reliable follow-up may need little or no laboratory testing. Prospective validation should examine missed serious diagnoses, test utilization, ED length of stay, return visits, patient-reported reassurance, and referral completion.

Table 2: Proposed PANIC-SAFE Framework (not yet validated).

Element	Action	Safety question
P — Primary stabilization	ABCDE, vital signs, SpO2, mental status; treat instability.	Is there an immediate physiologic threat?
A — Assess red flags	Define dominant syndrome, time course, risk factors, exposures, and pretest probability.	Which dangerous diagnoses remain plausible?
N — Necessary biomarkers	Select tests only when linked to a hypothesis and management threshold.	How will this result change care?
I — Immediate POCT	ECG, glucose, monitoring, blood gas, pregnancy test, and/or focused ultrasound as relevant.	Can a bedside test rapidly identify a reversible mimic or alternate diagnosis?
C — Clinical reassessment	Repeat vital signs/examination; reconcile response and discordant findings.	Does the entire presentation now make sense?
SAFE	Safe disposition, Advice, Follow-up, Explicit return precautions.	Is follow-up reliable and does the patient understand when to return?

Limitations and Research Priorities

This narrative review is limited by non-systematic study selection and by the indirectness of much of the evidence. Most diagnostic studies evaluate chest pain, dyspnea, or suspected PE populations rather than cohorts selected for suspected panic attack. Test performance therefore cannot be transferred uncritically to every psychiatric-appearing presentation. Technologies, assay thresholds, and local pathways also vary.

Research priorities include prospective ED cohorts using standardized panic assessments after medical evaluation; development and external validation of symptom-based pathways; evaluation of digital or bedside screening tools; and implementation studies that combine medical rule-out with rapid mental-health referral. Cost, radiation exposure, false-positive cascades, diagnostic equity, and patient experience should be measured alongside missed disease. The proposed PANIC-SAFE framework requires validation before clinical-policy use.

Conclusions

No biomarker confirms a panic attack. Safe emergency care depends on a risk-adapted process in which ECG, hs-cTn, D-dimer, glucose, blood gas analysis, lactate, electrolytes, endocrine or toxicologic tests, and POCUS are selected according to a specific clinical hypothesis. Validated pathways should be used in full, assay limitations respected, and discordant results reassessed. The goal is neither maximal testing nor rapid psychiatric labeling, but proportionate exclusion of time-critical disease followed by clear explanation, appropriate mental-health assessment, and reliable follow-up. PANIC-SAFE offers a structured sequence for that process and a testable model for future ED research.

Declarations

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Use of artificial intelligence: [Adapt to the target journal policy: disclose any language or editorial assistance as required; the author remains responsible for all content, citations, and clinical claims.]

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