

PSEUDOANGINA

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The Clinical Definition and Context of Pseudoangina

Pseudoangina represents a complex clinical phenomenon where an individual experiences chest pain identical in quality, distribution, and intensity to true **Angina Pectoris**, yet comprehensive medical evaluation reveals absolutely no underlying cardiac pathology or evidence of ischemic heart disease. This condition is fundamentally defined by the discordance between pronounced, often debilitating symptoms--such as crushing or constricting retrosternal pain--and the absence of objective medical findings that would typically confirm a cardiovascular etiology. The term itself, derived from the Greek prefix "pseudo" meaning false, precisely encapsulates the deceptive nature of the ailment, positioning it as a critical challenge in differential diagnosis for emergency room physicians and cardiologists alike. While the subjective suffering is undeniably real for the patient, the physiological origin lies outside the coronary arterial system, necessitating a shift in diagnostic focus toward non-cardiac sources, which often include musculoskeletal, gastrointestinal, or psychogenic factors.

Historically, the accurate identification of pseudoangina has been crucial, particularly in high-stakes medical environments where the immediate priority is ruling out a life-threatening myocardial infarction or unstable angina. The pain associated with pseudoangina can be so severe and textbook-like that initial presentation mandates immediate, intensive cardiac workups, including electrocardiograms (ECGs), cardiac enzyme assays (e.g., troponin levels), and stress testing. Only after these rigorous investigations consistently yield negative results and confirm a structurally and functionally sound heart can the diagnosis of pseudoangina be confidently applied. This process underscores a significant emotional burden for the patient, who is often terrified by the possibility of heart disease, and a diagnostic burden for the clinician, who must carefully navigate the fine line between unnecessary treatment and missed cardiac events, highlighting why detailed patient history and meticulous documentation are indispensable components of the initial assessment.

The distinction between true angina and pseudoangina is not merely academic; it dictates the entire course of management and prognosis. True Angina Pectoris signals underlying **coronary artery disease** (CAD), requiring interventions such as angioplasty, medication management (nitrates, beta-blockers), and aggressive risk factor modification to prevent future cardiac events. Conversely, pseudoangina, despite its alarming presentation, carries a far more benign cardiac prognosis; the patient's heart is, structurally and functionally, robust. However, the benign cardiac outlook does not imply a benign overall condition, as the recurrent pain often signals underlying issues related to anxiety, panic disorders, esophageal dysfunction, or rib cage abnormalities, demanding specialized non-cardiac therapeutic approaches to alleviate symptoms and improve long-term quality of life.

Furthermore, the example of individuals like Eric, who suffer intensely from chest pain even though their heart function is objectively verified as normal, serves as a poignant reminder of the powerful

connection between psychological distress and physical manifestation. Pseudoangina falls squarely into the domain of psychosomatic medicine, demonstrating how severe anxiety or panic attacks can trigger physiological responses--such as hyperventilation leading to chest wall muscle spasms or esophageal reflux mimicking cardiac pain--that are indistinguishable from true ischemic events. Recognizing this interplay is essential for the clinician, enabling them to transition the patient from the cardiac care pathway to appropriate psychological or gastroenterological consultation, ensuring the patient receives holistic and effective care tailored to the true source of their discomfort.

Symptom Presentation and Mimicry of Angina Pectoris

The cardinal feature of pseudoangina is its near-perfect imitation of the classic symptoms associated with myocardial ischemia, creating profound diagnostic confusion. Patients frequently report sensations of retrosternal pressure, heaviness, squeezing, or a crushing weight on the chest, often accompanied by radiating pain extending into the left shoulder, neck, jaw, or down the arm. These descriptions are highly characteristic of true **stable angina**, which is why initial triage protocols must treat every incidence of new-onset chest pain as cardiac until proven otherwise. However, subtle differences, often only discernible through detailed questioning and observation, can sometimes hint at a non-cardiac origin. For instance, pseudoanginal pain might last for hours or even days, whereas true ischemic pain rarely persists beyond 20 minutes without evolving into infarction, especially when resting or taking nitroglycerin.

While the classic description often dominates, pseudoangina frequently exhibits atypical features that assist in differentiation once cardiac causes are excluded. The pain is sometimes described as sharp, stabbing, or fleeting, lasting only a few seconds, which is highly inconsistent with ischemic pain that typically builds and subsides gradually. Furthermore, pseudoanginal discomfort is commonly localized to a very specific, small point on the chest wall (the "pointing sign"), and may often be reproduced or exacerbated by specific movements, changes in posture, deep breathing, or direct palpation of the chest wall or costochondral junctions, suggesting a musculoskeletal origin such as costochondritis or Tietze's syndrome. These features contrast sharply with true angina, which is typically diffuse and not positionally dependent, highlighting the necessity of a thorough physical examination focusing on the chest wall structures.

A significant subset of pseudoanginal presentations is closely linked to psychological stress, notably **panic disorder** and generalized anxiety. In these cases, the chest pain episode is often preceded or accompanied by overt symptoms of autonomic arousal, including hyperventilation, dizziness, tingling in the extremities (paresthesias), and intense fear or sense of impending doom. The hyperventilation associated with panic attacks can lead to respiratory alkalosis, causing spasms in the intercostal muscles and esophageal muscle, which directly contribute to the feeling of chest tightness and constriction, perfectly mimicking cardiac pain. Understanding this

physiological cascade--from psychological trigger to muscle spasm--is key to interpreting the patient's symptoms accurately, especially when the pain resolves quickly once the panic attack subsides or when the patient is reassured by negative cardiac test results.

Other non-cardiac systems frequently implicated in pseudoangina mimicry include the gastrointestinal tract, primarily through **gastroesophageal reflux disease** (GERD) and esophageal motility disorders. Severe acid reflux can cause intense, burning retrosternal pain that is often confused with angina. This esophageal pain, sometimes referred to as "esophageal angina," is particularly tricky because it can be exacerbated by lying flat and sometimes even relieved by nitroglycerin (due to its smooth muscle relaxant properties, affecting the esophagus as well as coronary arteries), blurring the diagnostic lines. Careful history taking regarding meals, swallowing difficulties (dysphagia), and the response to antacids often provides the crucial clues necessary to redirect the diagnostic inquiry away from the heart and toward the digestive system, thereby confirming a non-cardiac origin for the chest discomfort.

Etiological Factors: Psychological and Somatic Contributions

The etiology of pseudoangina is inherently multifactorial, usually stemming from a convergence of psychological vulnerabilities and identifiable somatic issues that are not cardiac in nature. On the psychological front, conditions such as chronic stress, major depressive disorder, and specific anxiety disorders--particularly panic disorder and illness anxiety disorder (hypochondriasis)--are heavily implicated. Patients prone to pseudoangina often exhibit a heightened state of physiological arousal and **catastrophic misinterpretation** of bodily sensations. A minor fluctuation in heart rate or a momentary chest ache, which a healthy individual would ignore, is immediately interpreted by the patient as a sign of imminent heart attack, thereby triggering a feedback loop of increasing anxiety and subsequent physical symptoms, perpetuating the painful cycle.

Somatic etiologies, independent of the psychological overlay, also account for a significant portion of pseudoangina cases. The chest wall itself is a frequent source of pain. Conditions like costochondritis, characterized by inflammation of the cartilage connecting the ribs to the sternum, or muscle strain in the intercostal or pectoral muscles, generate localized pain that can be intense and frightening. Since the chest wall pain originates in the somatic nervous system, it is often sharp, positional, and tender to the touch, qualities rarely associated with true cardiac ischemia. Furthermore, nerve entrapment syndromes in the thoracic spine or cervical spine can refer pain to the chest, compounding the mimicry and requiring specialized orthopedic or physical therapy evaluation to confirm the non-cardiac musculoskeletal origin.

The gastrointestinal system represents another major category of somatic contributors. Esophageal disorders, including diffuse esophageal spasm (a condition where the esophageal muscles contract abnormally) and severe, atypical GERD, are powerful mimics of cardiac pain.

Esophageal spasm, in particular, can cause intense, gripping pain that feels identical to ischemic pain because the esophagus and the heart share neural pathways (visceral afferents), leading the brain to misinterpret the source of the pain as cardiac. The diagnosis of these esophageal motility disorders often requires specialized testing, such as manometry, to measure the pressure and coordination of esophageal contractions, confirming the digestive system as the true source of the agonizing chest discomfort.

In many patients, the etiology is a complex interaction between these psychological and somatic factors. For example, a patient suffering from underlying GERD might experience mild reflux, but severe anxiety about their health amplifies the perception of this pain and triggers hyperventilation, leading to secondary muscular spasms. This complex interplay necessitates an integrated treatment approach. Understanding that Eric's pseudoangina, while not heart disease, might be rooted in highly sensitized pain pathways aggravated by chronic stress, shifts the focus from purely physical tests to comprehensive psychological assessment, pain management strategies, and targeted treatment of underlying physical conditions like **acid reflux** or postural issues.

The Critical Role of Differential Diagnosis

Differential diagnosis is arguably the most critical and challenging phase in the management of pseudoangina. Given the life-threatening implications of genuine Angina Pectoris, the primary objective is the rapid and definitive exclusion of cardiac ischemia. This process must be systematic and thorough, often beginning in the emergency department with immediate monitoring, administration of oxygen if necessary, and the rapid collection of objective data. The clinician must maintain a high index of suspicion for cardiac events, recognizing that even subtle symptoms can mask severe underlying coronary disease, meaning that the diagnosis of pseudoangina can only be made retrospectively, after all serious cardiac possibilities have been convincingly eliminated through established protocols.

The initial diagnostic battery typically includes a series of specific cardiac investigations. The 12-lead **electrocardiogram** (ECG) is essential to detect characteristic changes such as ST-segment elevation or depression, T-wave inversions, or pathological Q waves indicative of ischemia or prior infarction. Serial blood tests measuring cardiac biomarkers, particularly high-sensitivity troponin I or T, are crucial; elevated levels definitively confirm myocardial necrosis, ruling out pseudoangina. If these initial markers are negative, but suspicion remains high, further non-invasive testing is required. This often involves stress testing, either exercise-induced (treadmill) or pharmacological (dobutamine), coupled with imaging (echocardiography or nuclear scanning) to assess for inducible ischemia, which, if negative, greatly strengthens the case for a non-cardiac diagnosis.

Once the cardiac system is medically cleared, the focus shifts to non-cardiac possibilities. This involves a detailed exploration of the patient's history regarding meal timing, specific food triggers,

positional changes, and emotional stressors. The physical exam must rigorously check for point tenderness in the chest wall, reproduction of pain with movement (suggesting musculoskeletal involvement), and signs of hyperventilation or anxiety. If symptoms suggest gastrointestinal involvement, further specialized investigations may be required, including upper endoscopy (EGD) to visualize the esophagus and stomach lining, or ambulatory pH monitoring to quantify acid reflux episodes and correlate them with reported pain events, effectively isolating the digestive system as the source of the pseudoanginal symptoms.

The comprehensive nature of the differential diagnosis ensures that patients are not prematurely labelled with a psychological diagnosis when a physical, albeit non-cardiac, ailment exists. For example, confirmation of costochondritis through localized tenderness and lack of systemic inflammation, or confirmation of severe esophageal spasm via manometry, provides the patient with a concrete, physical explanation for their terrifying pain. This concrete diagnosis, even if non-cardiac, often provides significant psychological relief, reducing the anxiety that frequently contributes to the severity and recurrence of the pseudoanginal episodes, thereby breaking the cycle of medical uncertainty and symptom amplification.

Diagnostic Procedures and Exclusionary Criteria

The definitive diagnosis of pseudoangina is strictly a diagnosis of exclusion, contingent upon the consistent application and negative results of a standard set of cardiac investigations. The foundational exclusionary criteria are based on objective evidence demonstrating normal myocardial perfusion and function. The initial screening steps are designed to swiftly triage high-risk patients. These procedures include multiple serial measurements of cardiac enzymes over a period of several hours to rule out myocardial injury, alongside repeated **electrocardiograms** to monitor electrical stability and identify transient ischemic changes that might have been missed initially. If the patient presents during a pain episode, immediate documentation of the ECG during the discomfort is highly valuable, as the absence of ischemic changes at the peak of symptoms strongly argues against true angina.

Beyond the acute phase, non-invasive stress testing serves as the gold standard for ruling out inducible ischemia. Stress tests, whether exercise or pharmacological, aim to increase myocardial oxygen demand to challenge the coronary arteries. In a patient with pseudoangina, the stress test results must be unequivocally negative, showing no evidence of wall motion abnormalities (on echocardiography) or perfusion defects (on nuclear scanning). A functionally and structurally normal heart under conditions of maximum stress provides powerful objective evidence that the chest pain is not cardiac in origin. If atypical symptoms or high-risk factors persist despite negative non-invasive testing, the clinician may opt for coronary computed tomography angiography (CCTA) or even invasive coronary angiography, especially in older patients or those with multiple cardiovascular risk factors, to definitively visualize the coronary arteries and confirm the absence of

significant obstructive disease.

Once the cardiac system is cleared, the exclusionary criteria extend to other major organ systems. Specialized testing is deployed based on the patient's history. For potential gastrointestinal causes, upper endoscopy provides direct visualization of the mucosal lining, helping to diagnose erosive esophagitis or gastritis. Esophageal manometry measures the contractile function of the esophagus, confirming disorders such as achalasia or diffuse spasm, which are highly effective mimics of cardiac pain. For musculoskeletal causes, the exclusion criteria are primarily physical: the pain must be reproducible upon palpation of specific chest wall points, and diagnostic injections of local anesthetic (e.g., into the costochondral joint) should completely abolish the pain, confirming the localized, somatic source.

The integration of these negative findings allows the clinician to transition the patient confidently toward a non-cardiac diagnosis. The final diagnostic determination relies on a thorough synthesis of the negative cardiac results with positive findings from other systems, such as evidence of panic disorder (via psychological inventory) or functional pain mechanisms (e.g., chronic hyperalgesia). This rigorous process of elimination not only ensures patient safety but also provides the necessary authority to reassure the patient, a crucial step in treating pseudoangina, allowing them to shift their focus from fear of imminent death to managing the underlying physical or psychological condition responsible for the chronic and distressing symptoms.

Psychological and Pharmacological Management Strategies

Management of pseudoangina is primarily focused on addressing the underlying non-cardiac etiology, which frequently involves significant psychological intervention due to the high correlation between this condition and anxiety, panic, or somatization. Cognitive Behavioral Therapy (CBT) is often the cornerstone of psychological treatment. CBT aims to identify and modify the patient's catastrophic thought patterns regarding their bodily sensations. By teaching patients to recognize that their chest pain is a result of muscle tension or reflux rather than heart attack, the therapy gradually reduces the anxiety-symptom feedback loop. Techniques include exposure to feared internal sensations, relaxation training (such as controlled diaphragmatic breathing to counteract hyperventilation), and psychoeducation regarding the benign nature of the cardiac prognosis.

Pharmacological management varies significantly depending on the confirmed etiology. If anxiety or panic disorder is the principal driver, selective serotonin reuptake inhibitors (SSRIs) or serotonin-norepinephrine reuptake inhibitors (SNRIs) are often prescribed to reduce overall anxiety levels and decrease the frequency and intensity of panic attacks. These medications work by modulating neurotransmitter activity in the brain, thereby reducing the physiological hyper-reactivity that translates into physical symptoms. In cases where panic attacks are acute and debilitating, short-term use of benzodiazepines might be considered, although their addictive potential

mandates cautious and limited application, reserving them primarily for crisis intervention rather than long-term management of chronic pseudoangina.

When the pseudoangina is linked predominantly to gastrointestinal issues, pharmacological intervention targets the digestive system. Proton pump inhibitors (PPIs) are highly effective in reducing gastric acid production, treating GERD, and alleviating esophageal irritation that mimics cardiac pain. Prokinetic agents may be used if esophageal motility issues are confirmed, helping to normalize the speed and coordination of food passage through the esophagus. For musculoskeletal sources, treatment involves physical therapy, non-steroidal anti-inflammatory drugs (NSAIDs) for localized inflammation (e.g., costochondritis), and sometimes local anesthetic or corticosteroid injections to manage focal pain and inflammation in the chest wall joints, addressing the specific somatic trigger of the patient's discomfort.

A key component of effective management, regardless of the specific etiology, is physician validation and reassurance. Once the heart is cleared, the clinician must communicate the results clearly and confidently, validating the patient's experience of pain while firmly establishing the non-cardiac source. This reassurance is a powerful therapeutic tool, often referred to as the "therapeutic negative," which helps dismantle the fear of death that fuels the pseudoangina cycle. The integration of specialty care--involving cardiologists, gastroenterologists, physical therapists, and psychologists--ensures that all components of the complex condition are addressed, leading to better symptom control and significant improvements in the patient's overall psychological and physical functioning.

The Impact of Pseudoangina on Quality of Life

Despite its benign cardiac prognosis, pseudoangina can severely diminish a patient's quality of life, often leading to profound functional impairment and chronic disability. The recurrent episodes of intense chest pain, indistinguishable from a heart attack, generate persistent fear and anxiety. Patients frequently develop significant health-related worries, leading to repeated, costly, and unnecessary emergency room visits, a phenomenon known as "high utilization." This constant state of alert, coupled with the uncertainty surrounding the pain, contributes to debilitating chronic stress, potentially leading to social isolation as patients avoid situations or activities they fear might trigger an episode, significantly restricting their professional and personal lives.

The chronic nature of the pain and the lack of a definitive, easily understood diagnosis often result in patient frustration and feelings of being misunderstood or dismissed by healthcare providers. Patients may face skepticism regarding the authenticity of their pain, especially when medical tests repeatedly come back negative. This medical uncertainty can fuel **illness anxiety disorder**, where the patient becomes preoccupied with the fear of having an undiagnosed, serious illness, further amplifying their symptoms. This cycle of pain, fear, medical testing, and frustration creates a

profound psychological burden, often leading to secondary depression and generalized anxiety that requires dedicated psychiatric care alongside physical symptom management.

Furthermore, pseudoangina can dramatically impact physical function. Patients may mistakenly believe that physical exertion is dangerous, leading to severe deconditioning and avoidance behaviors. For instance, an individual who experiences chest tightness during exercise due to underlying anxiety may cease all physical activity, leading to weight gain, muscle atrophy, and increased risk for actual cardiovascular disease later in life. Breaking this cycle requires supervised physical activity programs and cardiac rehabilitation protocols tailored to non-cardiac patients, where the focus is on rebuilding confidence in the body's ability to exert itself without catastrophic consequences, a process that must be carefully managed to ensure psychological safety.

The economic impact is also substantial. High utilization of healthcare services, including repeated ambulance rides, emergency visits, and extensive diagnostic testing (ECGs, stress tests, CT scans), places a heavy financial burden on both the individual and the healthcare system. Lost productivity due to missed workdays and the need for long-term psychological therapy further contribute to the overall societal cost. Effective management, therefore, requires not only symptom relief but also a comprehensive strategy aimed at reducing healthcare utilization by providing robust psychological support and a clear, confirmed alternative diagnosis that the patient can understand and trust.

Prognosis and Long-Term Patient Education

The long-term prognosis for patients diagnosed with pseudoangina is excellent regarding cardiovascular mortality; these individuals are not at increased risk of heart attack or cardiac death due solely to this condition. This highly favorable cardiac prognosis is the most important piece of information to convey to the patient. However, the prognosis concerning symptom resolution is more variable and depends heavily on the underlying cause and the patient's adherence to specialized treatment protocols. If the pseudoangina is linked primarily to a treatable somatic condition, such as severe GERD or costochondritis, targeted medical or physical therapy interventions typically lead to significant symptom abatement. Conversely, if the etiology is rooted deeply in chronic panic disorder or somatization, symptom management may require long-term psychological and pharmacological commitment.

Long-term patient education is crucial for successful management and relapse prevention. Patients must be thoroughly educated about the specific, non-cardiac mechanism responsible for their pain—whether it is esophageal spasm, muscle tension, or hyperventilation-induced symptoms. Providing a tangible, understandable explanation helps demystify the frightening experience and empowers the patient to manage their condition proactively. Key educational points include recognizing early signs of escalating anxiety or reflux, understanding how stress directly impacts their physical pain

thresholds, and learning specific coping mechanisms, such as relaxation techniques or trigger avoidance, to interrupt the cycle before a full-blown pseudoanginal episode develops.

A structured approach to managing recurrent symptoms is also vital for long-term success. Patients should be taught to differentiate between their known pseudoangina symptoms and any new, genuinely concerning cardiac symptoms (though this can be challenging). This often involves creating an individualized action plan:

Step 1: Employ immediate coping strategies (e.g., controlled breathing, antacids).

Step 2: Evaluate situational triggers (e.g., high stress, specific foods).

Step 3: If symptoms persist or if new, truly alarming symptoms arise (e.g., profound shortness of breath unrelated to anxiety), seek medical attention.

This structured approach reduces reliance on unnecessary emergency services and fosters patient autonomy and self-efficacy in managing chronic, non-life-threatening pain.

Ultimately, effective long-term management of pseudoangina relies on maintaining a robust therapeutic alliance between the patient and a multidisciplinary care team. Regular follow-up appointments, even when the patient is asymptomatic, help reinforce the negative cardiac prognosis and allow for ongoing assessment of psychological health and somatic triggers. By shifting the narrative from "undiagnosed heart disease" to "manageable chronic non-cardiac pain," clinicians enable patients like Eric to regain control over their lives, drastically reducing the psychological distress and functional limitations imposed by this deceptive and debilitating condition. The focus remains on treating the person, not just the pain, recognizing the profound interaction between the mind and the body in the manifestation of persistent, non-cardiac chest discomfort.