

# PSEUDOANGINA

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## PSEUDOANGINA

**Primary Disciplinary Field(s):** Cardiology, Psychiatry, Gastroenterology, Internal Medicine

### 1. Core Definition and Clinical Presentation

Pseudoangina, often clinically categorized under the broader umbrella of **Non-Cardiac Chest Pain (NCCP)**, describes a syndrome where individuals experience acute chest discomfort that perfectly mimics the symptoms of true **Angina Pectoris**, yet lacks any demonstrable underlying coronary artery disease or myocardial ischemia. This distinction is crucial in medical diagnosis, as true angina is a cardinal symptom of potentially life-threatening cardiac conditions, demanding immediate intervention. Conversely, pseudoangina, while intensely distressing and often debilitating for the patient, arises from non-cardiac etiologies, ranging from gastrointestinal and musculoskeletal disorders to profound psychological distress. The defining characteristic is the disconnect between the severity of the subjective pain experience and the absence of pathological evidence of heart disease, confirmed through extensive diagnostic testing, including electrocardiograms (ECG), stress tests, and coronary angiography.

The clinical presentation of pseudoangina is highly variable, mirroring the spectrum of genuine cardiac pain. Patients frequently report substernal pressure, tightness, squeezing, or burning sensations, which may radiate to the left arm, jaw, back, or neck--classic features associated with myocardial ischemia. Because the symptoms are so convincing, initial medical response almost invariably focuses on ruling out acute coronary syndrome (ACS), leading to repeated emergency department visits and significant healthcare utilization. It is the failure to identify an organic cardiac cause after a thorough workup that shifts the diagnosis toward pseudoangina. This diagnostic shift often results in patient frustration, as they struggle to reconcile their undeniable physical pain with the medical conclusion that their heart is healthy.

Understanding pseudoangina requires acknowledging that the pain is fundamentally real, despite its non-cardiac origin. The mechanism of pain transduction and perception in NCCP is complex, often involving visceral hypersensitivity, autonomic dysfunction, or heightened pain awareness mediated by central nervous system factors. For instance, subtle esophageal spasms or inflammation, while not life-threatening, can generate intense sensory signals that the brain misinterprets as originating from the adjacent heart tissue. This reality underscores the challenge of managing the condition; clinicians must validate the patient's pain experience while simultaneously redirecting diagnostic and therapeutic efforts toward the true, underlying non-cardiac cause, which often involves a multidisciplinary approach encompassing gastroenterology, psychiatry, and pain management specialists.

## 2. Etymology and Historical Context of Non-Cardiac Chest Pain

The concept of chest pain mimicking cardiac disease without structural heart issues is not new, but its formalization and current clinical understanding have evolved significantly. Historically, before advanced diagnostic imaging, many cases of pseudoangina were simply classified as atypical chest pain or functional chest pain, often leading to misdiagnosis or, conversely, unwarranted anxiety regarding potential heart disease. The term **pseudoangina** itself reflects the deceptive nature of the symptom--a "false" angina. The widespread recognition of this condition gained prominence as diagnostic technologies improved, allowing physicians to definitively exclude cardiac ischemia as the cause in a substantial proportion of patients presenting with angina-like symptoms.

In contemporary medicine, the term Non-Cardiac Chest Pain (NCCP) has largely superseded **pseudoangina** in formal clinical settings, as NCCP provides a broader, more inclusive framework for diagnosis and treatment. NCCP is defined as recurrent chest pain similar to angina that remains unexplained after careful cardiac evaluation. Studies suggest that NCCP accounts for approximately 25% to 40% of all cases of chest pain evaluated in non-emergency settings, highlighting its substantial prevalence and clinical importance. The historical challenge was overcoming the initial bias that severe chest pain must equate to heart disease. The modern approach focuses instead on systematically investigating the numerous extracardiac systems that can generate comparable pain signals, recognizing the highly interconnected visceral sensory pathways in the thoracic cavity.

The historical development of treating this condition also reflects the growing understanding of the mind-body connection. Early conceptualizations sometimes dismissed non-cardiac chest pain as purely psychosomatic or hypochondriacal, leading to inadequate care. However, research in the late 20th and early 21st centuries firmly established physiological bases for many cases, particularly those linked to esophageal disorders (e.g., motility issues) and musculoskeletal problems (e.g., costochondritis). Simultaneously, the profound link between psychological states--specifically panic disorders and generalized anxiety--and the exacerbation or initiation of chest pain symptoms has solidified the role of psychiatric evaluation as an essential component of the comprehensive NCCP/pseudoangina diagnostic pathway. This evolution ensures that patients receive holistic care targeting both physiological and psychological stressors contributing to the pain experience.

## 3. Primary Etiologies and Pathophysiology

The etiologies of pseudoangina are diverse and typically categorized into three major domains: gastrointestinal, musculoskeletal, and psychological. **Gastrointestinal disorders** are the most common identified cause, with conditions affecting the esophagus leading the list. Key among

these is **Gastroesophageal Reflux Disease (GERD)**, where acid reflux irritates the esophageal lining, generating intense burning pain often misinterpreted as angina. Furthermore, esophageal motility disorders, such as diffuse esophageal spasm (DES) or nutcracker esophagus, involve uncoordinated or excessively forceful contractions of the esophageal muscle. These spasms can produce severe, constricting chest pain that is virtually indistinguishable from cardiac pain, sometimes even responding transiently to nitroglycerin, further complicating the diagnostic process.

The **musculoskeletal system** also serves as a significant source of pseudoangina. Pain originating from the chest wall, ribs, cartilage, or intercostal muscles is frequently sharp, localized, and reproducible upon palpation, which helps distinguish it from visceral cardiac pain. The most recognized musculoskeletal cause is **Costochondritis** (or Tietze's syndrome), an inflammation of the cartilage connecting the ribs to the sternum. Activities involving movement, specific postures, or deep breathing can exacerbate this pain, providing key diagnostic clues. Other musculoskeletal sources include muscle strain, vertebral issues, or nerve impingement in the thoracic spine. Identifying these causes often requires a careful physical examination, including the 'point tenderness' test, where reproducing the exact pain with pressure strongly indicates a chest wall origin rather than a deep, visceral source.

Finally, **psychological factors**, particularly severe anxiety, panic disorder, and somatization, play a crucial and often overlapping role. Panic attacks frequently include symptoms such as hyperventilation, shortness of breath, palpitations, and intense, crushing chest pain, leading patients directly to the emergency room fearing a heart attack. This pain is often exacerbated by the patient's catastrophic interpretation of their symptoms (fear of dying). In cases of chronic anxiety or somatization disorder, the patient may experience physical pain symptoms without a clear organic pathology, possibly due to a lowered pain threshold or heightened internal sensory monitoring. It is critical to note that the psychiatric etiology is often a diagnosis of exclusion and may coexist with subtle physiological changes, such as microvascular dysfunction or esophageal hypersensitivity, forming a complex interplay between mind and body that perpetuates the experience of chronic pseudoangina.

#### 4. Differential Diagnosis and Diagnostic Protocols

The diagnostic process for pseudoangina is primarily an exhaustive process of exclusion, designed to systematically rule out all serious cardiac causes before focusing on non-cardiac etiologies. The initial evaluation for any patient presenting with acute chest pain must adhere to protocols for diagnosing or ruling out Acute Coronary Syndrome (ACS). This involves a detailed history, physical examination, serial **Electrocardiography (ECG)**, and blood tests measuring cardiac biomarkers, such as troponin levels. If these initial screenings are negative for cardiac injury, further tests, including exercise stress tests, myocardial perfusion imaging, and potentially computed

tomography angiography (CTA) or standard coronary angiography, are utilized to confirm the absence of significant coronary artery obstruction or functional ischemia.

Once cardiac causes are definitively excluded, the focus shifts to the major non-cardiac systems. Diagnosis of gastrointestinal causes often involves specialized testing. A 24-hour esophageal pH monitoring or impedance testing can confirm GERD or detect non-acid reflux. High-resolution esophageal manometry is crucial for identifying motility disorders like DES or achalasia, which can generate significant chest pain. Endoscopy (EGD) may also be performed to visualize the esophageal lining for inflammation (esophagitis) or structural abnormalities, providing direct evidence for a GI cause. Accurate GI diagnosis is paramount because treatments for esophageal pain (e.g., proton pump inhibitors for GERD) are highly specific and effective when the source is correctly identified.

If both cardiac and major GI etiologies are ruled out, the physician investigates musculoskeletal and psychological causes. Musculoskeletal pseudoangina is usually confirmed clinically through the aforementioned physical examination maneuvers (reproducible pain upon palpation). Diagnosing the psychological component often requires structured screening tools, detailed patient interviews focusing on stressors, anxiety levels, and symptom amplification, and sometimes referral to a mental health professional. It is essential to avoid the pitfall of labeling the pain as "psychological" merely because organic causes are absent; rather, psychological factors must be actively identified and treated as a primary cause. The differential diagnosis, therefore, is a stepwise, iterative process requiring collaboration across multiple medical disciplines to ensure no potential underlying cause is overlooked.

## 5. Key Characteristics Differentiating Pseudoanginal Pain

While pseudoanginal pain can convincingly mimic true angina, detailed clinical history often reveals characteristics that help differentiate it from cardiac ischemia. One key differentiator is the relationship of the pain to exertion. True angina is classically precipitated by physical stress (e.g., walking uphill, intense exercise) and relieved promptly by rest or nitroglycerin. Pseudoangina, conversely, may occur at rest, often in the middle of the night, or may be unrelated to physical exertion, though pain related to esophageal spasm can sometimes be triggered by cold liquids or stress, which might confuse the picture. Pain lasting only a few seconds, or continuously for hours or days, is also highly suspicious for a non-cardiac origin, as typical ischemic angina rarely lasts beyond 20 minutes unless myocardial infarction is occurring.

Another distinguishing feature relates to the influence of specific activities or positions. Pseudoanginal pain, especially when musculoskeletal, is often sharp, localized to a small area, and may be altered by changes in posture, torso movement, or deep inhalation. For example, pain from costochondritis can be exacerbated by turning the body or pressing on the chest wall,

attributes that are inconsistent with deep, visceral cardiac pain. Gastrointestinal pain, such as that caused by reflux, frequently presents as a burning sensation and is often relieved by antacids or exacerbated immediately after eating or when lying flat.

Furthermore, the association with specific non-cardiac symptoms provides vital clues. Pain accompanied by dysphagia (difficulty swallowing), regurgitation, or chronic coughing strongly points toward an esophageal origin. Similarly, chest pain accompanied by symptoms of intense anxiety, panic, depersonalization, or hyperventilation suggests a primary psychological mechanism. While patient reporting can be subjective and sometimes inconsistent, a careful, structured history focusing on precipitants, duration, quality, location, and associated non-cardiac symptoms is often the most powerful tool for initially separating pseudoangina from genuine cardiac disease, guiding the subsequent, expensive, and time-consuming diagnostic workup.

## 6. Psychological Dimensions and Comorbidity

The relationship between psychological distress and pseudoangina is bidirectional and profound. For many patients, the chronic experience of chest pain, coupled with the initial fear of a heart attack and the subsequent medical uncertainty when tests return negative, leads to significant health anxiety, sometimes termed 'cardiac neurosis.' This anxiety, in turn, can heighten sympathetic nervous system activity, potentially leading to muscle tension, hypervigilance regarding bodily sensations, and even visceral hypersensitivity in the esophagus, thus exacerbating the pain cycle. The psychological suffering is often compounded by the feeling of being dismissed by the medical community when a definitive organic diagnosis is elusive.

In many instances, pseudoangina is a prominent manifestation of an underlying anxiety disorder, panic disorder, or depression. Panic attacks, characterized by sudden, intense surges of fear, trigger massive autonomic arousal, leading to physical symptoms like tachycardia, shortness of breath, dizziness, and, crucially, chest pain that is functionally indistinguishable from cardiac pain. When these episodes are recurrent, the patient begins to associate the physical sensation of chest pain with impending doom, leading to avoidance behaviors and significant impairment of daily life. The pain itself becomes a conditioning stimulus for fear, creating a vicious cycle where anxiety triggers pain, and the pain triggers more anxiety.

Effective management of pseudoangina must therefore address this psychological comorbidity. Cognitive Behavioral Therapy (CBT) has emerged as a cornerstone treatment, particularly when the pain is linked to anxiety or panic. CBT helps patients challenge catastrophic thought patterns related to the pain (e.g., "This chest pain means I am dying") and teaches coping mechanisms for managing anxiety and hyperventilation. Furthermore, treating underlying depression or generalized anxiety disorder with appropriate pharmacotherapy (such as Selective Serotonin Reuptake Inhibitors or SSRIs) can significantly reduce the frequency and intensity of pseudoanginal

episodes, demonstrating the critical role of central nervous system modulation in managing this pain syndrome.

## 7. Clinical Management and Treatment Strategies

Clinical management of pseudoangina begins with patient education and reassurance, particularly emphasizing the definitive exclusion of severe cardiac disease. This step is vital for reducing the patient's catastrophic thinking and breaking the cycle of anxiety-induced pain. The management strategy is then tailored based on the identified underlying non-cardiac etiology, necessitating highly specific, targeted treatments rather than a blanket approach.

For **gastrointestinal causes**, treatment protocols are clearly defined. GERD is primarily managed with lifestyle modifications (dietary changes, weight loss, elevation of the head of the bed) and pharmacological intervention, typically involving long-term use of **Proton Pump Inhibitors (PPIs)**. If esophageal motility disorders are identified, treatments may involve calcium channel blockers or nitrates to relax the esophageal smooth muscle, or, in more refractory cases, psychological interventions aimed at reducing visceral hypersensitivity. For cases where no specific GI pathology is found but visceral hypersensitivity is suspected, low-dose neuromodulators, such as tricyclic antidepressants (TCAs), have shown efficacy in reducing pain perception by modulating central and peripheral pain pathways.

If the etiology is primarily **musculoskeletal**, treatment focuses on inflammation reduction and physical therapy. Nonsteroidal anti-inflammatory drugs (NSAIDs) are often used for conditions like costochondritis. Targeted physical therapy, stretching exercises, and sometimes local corticosteroid injections can alleviate chronic chest wall pain. When the pain is rooted in **psychological distress**, the primary therapeutic modalities shift to psychological intervention. As previously noted, CBT and relaxation techniques are highly effective. Furthermore, psychiatric medications, including SSRIs or SNRIs, are often beneficial in reducing the frequency of panic attacks and associated chest pain, allowing the patient to regain control over their symptoms and improve their overall quality of life. The comprehensive management of pseudoangina requires integrating these specialized treatments under the coordination of a primary care physician or internist who can facilitate referrals and monitor progress across disciplines.

### Further Reading

[Wikipedia: Non-cardiac chest pain](#)

[Mayo Clinic: Gastroesophageal Reflux Disease \(GERD\)](#)

[Wikipedia: Angina pectoris](#)