ATYPICAL SYMPTOMS OF ACUTE CORONARY SYNDROME IN WOMEN: INCREASING NURSE PRACTITIONERS’ AWARENESS IN PRIMARY CARE SETTINGS

By

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Atypical Symptoms

ATYPICAL SYMPTOMS OF ACUTE CORONARY SYNDROME IN WOMEN: INCREASING NURSE PRACTITIONERS’ AWARENESS IN PRIMARY CARE SETTINGS

Abstract

Chair: Lorrie Dawson

The leading cause of death for women in the United States is due to coronary artery disease (CAD). One of the main sequelae of CAD is acute coronary syndrome (ACS). Evidence is clear that women may experience atypical symptoms of ACS more than men. This article provides a review of ACS, typical, and atypical symptoms. It provides a description of populations more likely to present with atypical symptoms. The article continues by suggesting strategies nurse practitioners in primary care can use to include ACS as a diagnostic differential, when, and how to send a patient for additional diagnostics and observation. Research has demonstrated women who present with atypical symptoms ACS are underdiagnosed and undertreated and it is essential this population receive more attention and education.
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Imagine the fictional case of L.H., a 69 year-old woman with a history of diabetes, chronic hypertension, and mild obesity who presents to the clinic nurse practitioner with symptoms of weakness, shortness of breath, and persistent nausea for the past three days. She put off coming in because she dismissed the symptoms as “just being a bit more tired that usual.” After waiting an hour in the lobby she is finally in front of the provider discussing her symptoms. Subsequent to a lengthy assessment, she is transported to the hospital where two days later is diagnosed with acute coronary syndrome for an evolving myocardial infarction. Within a month she readmitted to the hospital following complications of congestive heart failure secondary to coronary artery disease. The case of L.H. evokes the question, “Could this sequelae of events been prevented by primary care providers?”

急性冠状动脉综合征 (ACS) 描述了一组需要立即注意和干预的冠状动脉诊断。急性心肌病是美国妇女的主要死因。自1984年以来，更多的妇女死于心血管疾病。一种误解，即“心脏病是男性疾病”在2005年的一项调查中反映了36%的女性并不认为她们有心血管疾病的风险。胸痛是提供者用来诊断ACS的主要症状。然而，越来越多的证据表明，ACS在女性中可能表现为没有经典的胸痛或以被认为不典型的心肌缺血症状表现。研究已表明，女性更可能将她们的症状归因于其他原因，等待更长的时间进行评估，并接受比男性报告胸痛并被诊断为ACS的女性更少的干预措施。

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The purpose of this article is to provide information to primary care providers regarding atypical ACS symptoms that should create an increased index of suspicion among female patients. This article serves to encourage health care providers to educate at risk patients and families to recognize and act upon potential atypical symptoms of ACS in women.
Overview of Acute Coronary Syndrome

Definition of Acute Coronary Syndrome

The triad of conditions that comprise ACS are unstable angina (UA), non-ST segment elevation myocardial infarction (NSTEMI), and ST segment elevation myocardial infarction (STEMI).\textsuperscript{1-3,21} STEMI describes the condition of transmural damage to the myocardium secondary to ischemic myocardial cellular necrosis.\textsuperscript{21} A STEMI can be diagnosed with observation of new elevation of the ST segment at the J-point on electrocardiogram (EKG) tracing using the parameters of $\geq 1.5$ millivolts (mv) in women or $\geq 2$ mv in men seen in two or more physiologically contiguous leads in the absence of left bundle branch block (LBBB) or left ventricular hypertrophy (LVH).\textsuperscript{22,23} NSTEMI describes myocardial ischemia and/or infarction with ST depression or marked T-wave inversion and/or elevated cardiac biomarkers.\textsuperscript{1,21} UA is clinically similar in cause and presentation to NSTEMI but has no collectively agreed definition.\textsuperscript{2,11,17} Existing literature places it amid the continuum between myocardial infarction (MI) and stable angina.\textsuperscript{2,11,17,21}

The 2007 revision of the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Practice Guidelines makes little differentiation of UA and NSTEMI on initial presentation.\textsuperscript{17} The ACC/AHA identifies myocardial ischemia as NSTEMI when enough cellular damage has occurred to cause cardiac biomarkers to be detectable, thus differentiating UA and NSTEMI diagnoses via degrees of severity.\textsuperscript{17}
Pathophysiology of ACS

ACS is the direct consequence of cellular ischemia of myocardial tissues.\textsuperscript{2,17,21,22} The pathogenesis of myocardial ischemia begins with the development of atherosclerotic plaques within the coronary arteries through the deposition of lipid-rich foam cells.\textsuperscript{2,17,21} Ischemia occurs either when the plaque completely occludes the lumen of the vessel or more commonly, when a plaque ruptures.\textsuperscript{2,17,22} Rupture can be caused by one or a combination of several mechanisms but most common is arterial inflammation from non-infectious sources which destabilize the plaque.\textsuperscript{17} This rupture activates focal deposits of thrombin and fibrin which in turn cause platelet aggregation.\textsuperscript{2,17} The rupture of plaque and subsequent formation of a partially or fully occlusive thrombus decreases blood supply to cardiac tissues creating a perfusion imbalance where demand exceeds supply.\textsuperscript{2,17,21,22}

Less common causes of coronary vascular lumen narrowing causing ischemia include vasospasm, dissection, and aneurisms.\textsuperscript{17} Myocardial cell ischemia if prolonged, will lead to cellular death.\textsuperscript{2,17,21,22} Cellular death can begin as soon as 20 minutes after the ischemia begins.\textsuperscript{22} Depending on perfusion deficit, myocardial tissue demand, and conditioning, ischemic conditions continuing beyond two hours can cause tissues within the affected zone to have significant enough necrosis as to be detectable on post mortem exam.\textsuperscript{22}

When myocardial tissues begin to destabilize and necrose, cellular contents spill into the circulating blood stream.\textsuperscript{17} These biomarkers include several types of troponin, creatinine kinase, elevated brain natriuretic peptide (BNP) and C-reactive protein (CRP). Troponin I (cTnl) is a protein that binds the troponin complex to the cardiac
myofilaments.\textsuperscript{24,25} Troponin T (cTnT) is another cardiac-specific protein which binds the troponin complex to tropomyosin.\textsuperscript{24,25} Creatinine kinase (CK) is an enzyme found primarily within skeletal and cardiac muscle tissues.\textsuperscript{25} MB is the cardiac specific CK isoenzyme.\textsuperscript{25} These biomarkers along with myoglobin levels give indirect evidence of cardiac tissue damage and timing of event.\textsuperscript{17,25} When patients experience UA and NSTEMI there is gender variation in the release cardiac biomarkers with females more prone to express elevated BNP and CRP while less frequently exhibiting elevated CK-MB and troponins.\textsuperscript{11}

\textit{Gender Differences in ACS Pathophysiology}

Gender differences exist affecting the pathophysiological factors causing ACS.\textsuperscript{24,26} One of the most obvious is the influence of female sex hormones influence which appear to slow development of coronary artery disease (CAD) in premenopausal women.\textsuperscript{14,21,24,26} Research suggests even at the molecular level, women’s cellular receptors react differently to similar levels of circulating hormones than men.\textsuperscript{14} Endogenously produced estrogen in women stimulates enhanced endothelial function and arterial dilation.\textsuperscript{14} This influence rapidly diminishes after menopause.\textsuperscript{14,24} Women’s CAD risk levels equal to those of men within 10 years post-menopause.\textsuperscript{14,24} This coincides with evidence in which the average age of a woman presenting with ACS generally manifests 10 years later than men with ACS.\textsuperscript{26,27}

Vascular abnormalities appear at a statistically higher frequency in women than men.\textsuperscript{24,26,28} The coronary arteries in women have been found to be smaller, more prone to certain plaque formations, and less compliant.\textsuperscript{24,26,28} Even when body mass differences are taken into account, cardiac stroke volume in women is 10\% less than men.\textsuperscript{14}
Atypical Symptoms

Cardinal Symptom of ACS

The principal symptom in patients presenting with ACS is chest pain.\textsuperscript{1,9,27,29} Current research indicates that nearly 70\% of patients diagnosed with ACS present reporting a primary complaint of chest pain.\textsuperscript{1} The nature, quality, timing of the chest pain varies amongst existing research and protocols. In the 2007 revision, the ACC/AHA identified typical symptoms of cardiac ischemia as pain or discomfort radiating to the upper abdomen, jaw, chest, upper arms generally lasting longer than twenty minutes not affected by movement or repositioning.\textsuperscript{22} The pain/discomfort can be area specific or disseminated through the aforementioned areas.

Atypical Symptoms of ACS

There is no universal agreement on specific atypical symptoms of ACS among experts.\textsuperscript{10,14} Literature generally portrays atypical symptoms as those experienced in the presence of myocardial ischemia but lacking accompanying expected chest pain.\textsuperscript{1,3,10,14} Atypical symptoms of ACS are identified as those not falling into the expected cluster of presentations commonly associated with myocardial ischemia i.e. symptoms occurring in the absence of chest pain.\textsuperscript{3,10} Nearly a third of all patients receive a final diagnosis of ACS or one of the singular diagnoses it encompasses but do not present to HCPs with chest pain as a primary complaint.\textsuperscript{1,10} UA and NSTEMI comprise about 67\% of the ACS diagnoses without chest pain.\textsuperscript{3} The list of atypical symptoms mentioned in current literature is exhaustive. Common atypical symptoms include dyspnea, diaphoresis, fatigue, weakness, malaise, syncope, dizziness, nausea/vomiting, and abdominal pain.\textsuperscript{1,3,10,13,14,19,27} Atypical symptoms have been correlated and observed in patients
mislabeled with a diagnosis other than ACS. These symptoms can confound an accurate diagnosis and lead to potentially adverse outcomes for patients.

**Populations More Likely to Present with Atypical Symptoms of ACS**

Mounting research indicates within the population of patients diagnosed with ACS, exist specific risk factors, increasing the likelihood of experiencing atypical symptoms during myocardial ischemia. These risk factors include advanced age, being female with concurrent comorbidities of diabetes, hypercholesterolemia, hypertension, and/or congestive heart failure (CHF). Atypical symptoms were statistically less common in patients who smoked or had a known history of CAD.

Data compiled from the Global Registry of Acute Coronary Events (GRACE) demonstrated patients not experiencing chest pain during an ischemic event were likely greater than 75 years of age and female. There is little evidence to explain this phenomenon although some research suggests that the aging process results in decreased awareness to noxious stimulation during ischemia.

Women presenting with ACS more frequently report atypical symptoms than their male counterparts. Women are less likely to describe chest symptoms as “pain” than men. This difference may explained by psychological differences between men and women. Women are three times more likely to have anxiety/depression syndromes which can mimic ischemic coronary symptoms.

Diabetes is often mentioned as an independent factor of painless ACS. Research suggests an explanation for this phenomenon. Peripheral neuropathy is a common sequelae of chronic diabetes disease process and investigators suggest some
patients may develop cardiac neuropathy thus diminishing ischemic nocioception, preventing perception of chest “pain”.¹

Congestive heart failure is another comorbidity associated with the presentation of atypical symptoms of ACS.¹,³,¹⁰,²⁴ The greater the severity of the CHF (Killip class) of the patient, the greater the likelihood atypical symptoms and of absence of chest pain as a chief complaint.¹ One theory attempting to explain this trend suggests a “blunting” of perception to pain which is pre-existing and heart failure symptoms may be the only indicators of ischemia.¹

Additional Factors Causing Delay in Care with Women with ACS

When female patients present to HCPs with atypical symptoms of ACS, they stand a statistically greater chance of experiencing delays in transport, diagnosis, and treatment.¹⁵⁻¹⁸ Studies also indicate mortality and morbidity rates associated with ACS in women are 15% to 20% greater than men.⁴

Women may be partly responsible for delays in initiating care when faced with symptoms not commonly associated with ACS.²⁶ A significant percentage of women may not even perceive themselves to be at risk for cardiovascular disease.³¹ The theoretical concept of Illness Representation is a central construct in Leventhal’s Self-Regulation Theory may serve to explain this behavior.³² This construct states there are parallel processing mechanism of cognitive and emotion that factor attributes of timeline, symptoms, consequences, cause, cure, and the coherence of the patient’s thought process on the illness.³² Literature provides examples of women using coping mechanisms like minimization and adopting a “wait and see” attitude and even self-management as factors
to explain potential delays diagnosis and treatment of ACS.\textsuperscript{4,19,26} Women also tend to try to contact their own providers during ACS events more often than men.\textsuperscript{15}

Primary care providers (PCP) also contribute to delays in patients receiving appropriate care during ACS events.\textsuperscript{15,33} The average wait time to see a PCP is nearly an hour and a half.\textsuperscript{15} Chief complaints atypical for ACS can cause a delay in PCP recognition and diagnosis of the emergent situation and on average it takes a PCP 16 minutes longer to evaluate a woman than a man.\textsuperscript{15}

Research demonstrates emergency medical systems (EMS) and emergency departments (EDs) add to delays in care and diminished intensity in which care is delivered to women with atypical ACS symptoms.\textsuperscript{18} Prehospital care by EMS transport (ambulance) is protocol and algorithm driven.\textsuperscript{18} Despite protocols, women appear to receive less interventions like aspirin and intravenous (IV) access during ACS events.\textsuperscript{18} Even when women’s complaints were similar to men’s, providers on the ambulances statistically appeared to favor a less aggressive approach.\textsuperscript{18} In EDs, women were less likely to have an EKG within 10 minutes of their arrival despite similar reported symptoms as men.\textsuperscript{4} Evidence indicates less aggressive employment of medications, thrombolytics, and percutaneous interventions even after a diagnosis of ACS is determined.\textsuperscript{4,18} Employment of interventions in women were more likely to take longer to initiate than their male counterparts.\textsuperscript{4}

There is no singular causative factor that explains gender differences in morbidity or mortality. The increased prevalence of comorbidities in women of hypertension, diabetes, congestive heart failure, and greater age may contribute to this inequity but no definitive studies exist to confirm or deny this theory.\textsuperscript{4,10,26}
Diagnosing Atypical ACS in the Primary Care Setting

The settings in which PCPs see patients vary extensively. They can range from a tiny office in a strip-mall with only the basic tools of assessment to large clinics located directly on hospital campuses with access to cardiac catheterization suites. Cognitive assessment tools such as history taking, physical examination, and provider awareness are available regardless of setting. Diagnostic testing such as EKGs and serum cardiac biomarkers are valuable and necessary in confirmation of a diagnosis of ACS, but may not always be immediately available to the PCP.11,34-36

One of the most important tools available to HCPs is collection of a rapid and accurate history.11,36 HCPs should be aware of potential biases while collecting information and avoid “diagnostic misattribution” with women.29,37 Providers may find with numerous co-illnesses, characteristics of a new illness may be attributed to existing diagnoses.29 A review of multiple studies illustrated erroneous diagnostic bias by HCPs of women presenting with atypical symptoms contributed to missed or delayed diagnoses of ACS.29

The ACC/AHA has identified five factors associated with ACS aiding in risk stratification of a patient.35,36 They follow in order of diagnostic importance: female beyond age 65 years or male greater than 55 years; character of the chest pain; previous diagnosis of CAD; gender; number of classic risk factors present.36 Various sources imply chest pain with specific qualities and locations have independent diagnostic
potential for CAD and ACS. The ACC/AHA guidelines suggest simply the presence of chest discomfort/pain be used to elevate the index of suspicion of ACS.

Physical examination of the patient can also assist in narrowing the list of potential differential diagnoses. Auscultation of transitory mitral valve regurgitation, rales or indications of pulmonary edema, diminished blood pressure, tachycardia, and diaphoresis have been associated with cardiogenic shock and necessitate a high suspicion of ACS requiring emergent interventions. A recent study of clinical features used to identify MI revealed active diaphoresis in the presence of the HCP was found to be the strongest predictor of MI. This phenomenon is explained by implication that diminished cardiac output and sympathetic nervous system response to discomfort can cause diaphoresis. Multiple studies indicate diaphoresis during ACS is more commonly reported by and observed in men.

Cardiac serum biomarkers can determine whether damage has occurred to myocardial tissues. These biomarkers vary as to timing of detection, peak, and duration. As with EKGS, other medical conditions may be the root cause of elevated cardiac biomarkers. These include but are not limited to recent cardiac surgery, rhabdomyolysis, renal failure, drug toxicity, burns, or myocarditis.

Research-based guidelines and algorithms have been employed with varying degrees of success in risk stratification and diagnosing ACS. These instruments are valuable aids which can aid HCPs in clinical decision-making. These tools cannot substitute entirely for HCP judgment because they are unable to process additional subtle factors used in decision making. Most ACS algorithms and guidelines give substantial weight to chest pain as a major predictor and guide to interventions.
employed. The most recent revision of the ACC/AHA guidelines on UA/NSTEMI added subsections on gender differences in detection of ACS.

Implications for Nurse Practitioners in Primary Care

Nurse practitioners (NPs) are a rapidly growing presence in the primary care setting. It is estimated that approximately 85% of NPs are trained in primary care of patients. NPs work in a wide variety of settings ranging from convenience care clinics to inpatient hospital settings. With the number of medical school graduates entering primary care residencies are dwindling, NPs find themselves in the position of PCP for an increasing number of patients.

Challenges Faced in Primary Care Settings

As with the case study of L.H., women with atypical presentations of ACS in primary care encounter a host of diagnostic and intervention difficulties and for both provider and patient. These include potentially remote location of clinics, EMS presence and transport times, availability of diagnostic instruments, treatment options, and limited provider and patient awareness and education.

PCPs’ skill competency and practice environment contribute to their ability to rapidly and accurately recognize atypical ACS presentation. Women going to their PCPs in general, wait longer to be diagnosed correctly, begin receiving treatment, and transport to appropriate facilities. This is exacerbated when presenting with atypical symptoms of ACS similar to those of L.H. EKG and rapid laboratory results evaluating serum cardiac biomarkers may not be available to the NP and subsequently be unable to aid in making a definitive diagnosis of ACS. Even if these tools are available, initial results may be non-diagnostic.
Atypical Symptoms

Women are often not educated to risks and symptoms associated with ACS. As a result, there may occur a misattribution or downplaying of symptoms of ACS to other causes. Women also tend to call their PCPs more than men during ACS. They may also delay or not request EMS transport because of a misunderstanding of appropriate use of these providers. HCPs agree through education, there is a positive affect on patients like L.H. future actions in seeking immediate care for suspected ACS. There is disagreement where this education should occur. Most physicians feel primary care has greater opportunities for “teachable moments”. With diminished face to face time with patients, PCPs are faced with the challenge of educating patients like L.H.

*When to Send a Patient to the Hospital*

When L.H. presented to the clinic with signs and symptoms of ACS, the nature of primary care implies her condition necessitated emergent and extended care beyond the scope of practice and length of a standard clinic visit. This population benefits from being transferred to the care of a hospital or chest pain center capable of dealing with possible sequelae seen in myocardial ischemia. In the presence of signs and symptoms of ACS, lack of or low likelihood of traditional risk factors should not dissuade providers from patient referral to an ED. (see ACS Algorithm)

The ACC/AHA guidelines of 2007 recommend initial assessments not occur via phone conversation. Women calling PCPs reporting symptoms suspicious for ACS should be evaluated at either an ED or acute care facility in which EKG and serum biomarkers can be rapidly evaluated by a physician. PCPs can have an opportunity to impart an increased sense of urgency to patients like L.H. and her family knowing
women demonstrate an increased prevalence toward atypical presentation of symptoms of ACS and a tendency to delay calling EMS.\textsuperscript{15,43}

Had L.H. called when she was experiencing her symptoms the PCP could have instructed her to call 9-1-1 especially if experiencing sudden symptoms of weakness, shortness of breath, chest pain, nausea, tachycardia, or diaphoresis suggesting ACS.\textsuperscript{17} L.H. was experiencing sudden symptoms of cardiogenic failure or myocardial ischemia at rest lasting greater than 20 minutes, and should have been initially evaluated at an ED.\textsuperscript{36} Transportation via private vehicle by relatives or friends should be discouraged.\textsuperscript{17} Research suggests private transport of patients where calling EMS transport would delay care more than 30 minutes.\textsuperscript{11}

\textit{Interventions to Consider Prior to Transport}

If a woman presents to her PCP with atypical symptoms suspicious for ACS such as in the case of L.H., interventions in clinical settings should not delay EMS transport to an ED.\textsuperscript{17} L.H. could benefit from relatively quick PCP interventions such as oral medications, supplemental oxygen, and IV access can be safely administered in a clinical environment if available.\textsuperscript{17,36} If rapidly available, additional diagnostic procedures for ACS patients may include an EKG, chest x-ray, and rapid blood test results.

\textit{Transfer of Information and Patient Care}

One dilemma faced by EDs when attempting to initiate patient care are provision of accurate information.\textsuperscript{46} Lack of pertinent patient history and information can increase time required to effectively diagnose and initiate patient treatment.\textsuperscript{46} There is no singular database by which a HCP may access any patient’s health information. Until this
mechanism exists, EDs rely on PCPs and EMS to forward patient health histories and chart information to them.\textsuperscript{46}

L.H.'s information such as health history and previous EKGs are of great diagnostic value to HCPs in the EDs and acute care environment when evaluating patients with symptoms suspicious for ACS.\textsuperscript{44} Additional information such as interventions initiated and effects on L.H. should be included. Copies of the chart, current medication list, and test results may be faxed to receiving facilities or hand-carried with the patient by EMS personnel. A courtesy call to the receiving facility by the PCP or PCPs representative can provide information and advance notification of L.H.'s pending arrival.

Implications for Research

To date, there are no definitive studies tracking how many NPs in the role of a PCP encounter women having atypical symptoms of ACS, nor what interventions and actions are taken during these interactions. There is little to no mention in literature regarding disruptions in the primary care environment to both provider and patient when ACS is a suspicion and EMS is activated. Further investigation may aid in development of potential algorithms that PCPs could employ to recognize and initiate earlier patient care, especially with women. Additional research might demonstrate differences in patient teaching effectiveness regarding atypical ACS symptoms by NPs versus other providers.

Conclusions

NPs are rapidly becoming the new stewards of primary care. As NPs become evermore present, so too do the chances of encountering female patients presenting with
atypical symptoms of ACS. Given the knowledge atypical symptoms may be more
typical for this subpopulation and these women may not be aware of their risks for ACS
NP’s can consider ACS more often as a differential diagnosis. Women with atypical
symptoms of ACS face higher mortality and morbidity than their male counterparts, the
imbalance may be alleviated in part with increased awareness and education for both
provider and patient.
Atypical Symptoms

Suspected Atypical ACS PCP Response Algorithm

Patient calls PCP via phone

Female patient with risk factors of DM, CHF, age >70 reporting no chest pain but recent or new onset sx suggesting atypical ACS.

Have pt call 9-1-1 for transport to ED for evaluation

Call anticipated receiving facility to notify of patients pending arrival and fax records if possible

Female patient presents at the clinic to PCP

Pt reports sx of recent/new onset of dyspnea, weakness, fatigue, near syncope/syncope, nausea/vomiting, diaphoresis, without typical ACS chest pain.

Symptoms unrelieved for >20min at rest or s/s of hemodynamic instability?

no

Continue assessment but consider atypical ACS presentation as a lower probability differential

yes

Does the pt have risk factors for increased risk of atypical ACS like diabetes, CHF, HTN, age 70 or greater?

no

yes

Call for EMS transport to ED for evaluation for ACS

Unless contraindicated, give chewable ASA, keep pt at rest, make copies of chart, notes, and records, call anticipated receiving facility for report. If time allows, EKG, supplemental oxygen via nasocanula, and NTG unless hemodynamic instability contraindicates administration.

Flow chart adapted from Angina/NonST-Elevation Myocardial Infarction ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Myocardial Infarction
Table 1

Additional Differential Diagnoses with Similar Symptoms as Atypical ACS

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>Shortness of breath, chest discomfort, fatigue, weakness, epigastric discomfort, nausea/vomiting, diaphoresis, rales</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Fatigue, epigastric pain, shortness of breath, palpitations</td>
</tr>
<tr>
<td>GERD</td>
<td>Epigastric discomfort, shortness of breath, nausea/vomiting</td>
</tr>
<tr>
<td>Esophageal spasm</td>
<td>Epigastric discomfort/pain, nausea</td>
</tr>
<tr>
<td>Costochondritis</td>
<td>Chest discomfort, shortness of breath, fatigue</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Chest discomfort, shortness of breath, diaphoresis, palpitations, weakness, syncope</td>
</tr>
<tr>
<td>CHF</td>
<td>Shortness of breath, chest discomfort, edema, rales, fatigue, weakness, tachycardia</td>
</tr>
</tbody>
</table>

Adapted from Dumphy, *Primary Care: The Art and Science of Advanced Practice Nursing*, 2007
References


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41. American College of Nurse Practitioners. What is a NP?


